

Fig 1 Pigeon K207 Most ataxic, the head and neck twisted and turned backward, the lower part of the body braced on the floor, legs stretched laterally and forward, phalanges of toes flexed resulting in hollow foot

Fig 2 Pigeon K137 Body swaying forward with flapping wings The wings and tail are shortened

Fig 3 Pigeon K172 The tail and wings are short, owing to frequent use as supports of the trunk, the feathers are worn down and appear as if they had been cut off with scissors

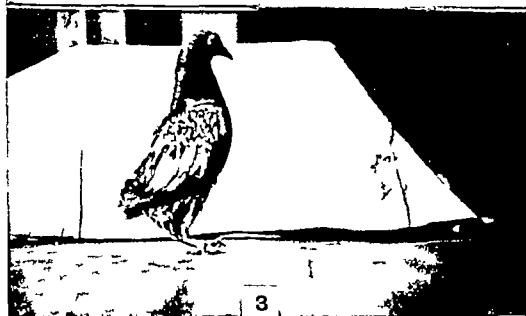
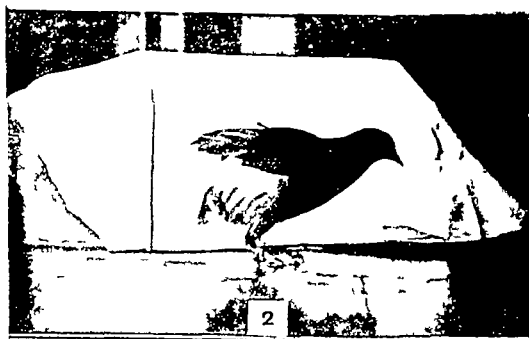


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Winkler, Dogiel, Munzer and Wiener, Boyce and Warrington, Murphy, Ziehen, Williams and Brouwer) It is not easy, therefore, for any one to study accurately any changes that have occurred in the nervous system of the pigeon For this reason each section of the affected birds was treated quite the same way as a corresponding control section This not only gives us a comparison with the normal structure, but also serves to show us any artefacts that may be present

The birds were narcotized with ether, while I opened the cranial cavity and spinal canal to take out the whole brain and spinal cord During the time of the removal the brain and cord were both rinsed in physiological salt solution, and before fixation, measured and weighed The brain stem, cerebellum, and the spinal cord of both the normal and affected birds were used for microscopic examination The brain was cut through proximally at the level of the posterior third of the optic lobes and distally at a point separating the medulla oblongata from the spinal cord Half of the cerebellum was left attached to the medulla and the whole fixed in 10 per cent neutral formalin solution, while one portion of the half of the cerebellum removed was fixed in Zenker's formalin solution and the other in alcohol The following is the formula for Zenker's formalin solution used

Bichromate of potassium	2 5 grams
Bichromate of mercury	5 grams
Sodium sulphate	1 gram
Formalin	10 cc
Distilled water	100 cc

From the spinal cord two parts were taken, one from the cervical region and the other from the lumbar region Each piece was cut into three divisions, one put in Zenker's formalin, one in 10 per cent neutral formalin, and the third in alcohol Each specimen had a control piece from the normal bird and both were treated the same way in the same bottle After fixation, regular dehydration followed with both pathological and control specimens Both pieces were imbedded in the same block of paraffin and cut with the microtome at the same time With each stroke of the blade, then, two sections would be made, one pathological

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TABLE 1  
*Weight of brain and spinal cord*

	K137 (A)	K187 (N)	K153 (A)	B173 (N)	K207 (A)	K199 (N)	K172 (A)	AVERAGE NORMAL	AVERAGE AFFECTED
Sex	$\sigma^1$	$\sigma^1$	$\phi$	$\sigma^1$	$\phi$	$\sigma^1$	$\sigma^1$		
Age (days)	300	228	246	424	220	232	230	293	248
Body weight (grams)	343	371	315	353	318	364	339	364	329
Weight of whole brain	1 833	2 004	1 699	1 987	1 686	1 941	1 840	1 977	1 763
Weight of brain and spinal cord	2 515	2 801	2 289	2 722	2 342	2 752	2 528	2 758	2 419
Weight of spinal cord	0 682	0 797	0 590	0 735	0 656	0 811	0 688	0 781	0 654
Weight of the proximal portion of brain <sup>1</sup>	1 220	1 262	1 158	1 295	1 155	1 222	1 270	1 257	1 201
Weight of the distal portion of brain <sup>1</sup>	0 613	0 742	0 541	0 692	0 535	0 719	0 589	0 718	0 570
Ratio weight of brain and cord to body weight	1 135 3	1 132 4	1 137 6	1 131 5	1 135 7	1 132 3	1 134 0	1 132 0	1 135 9
Ratio weight of the distal portion of brain to whole brain	1 2 990	1 2 687	1 3 142	1 2 871	1 3 153	1 2 699	1 3 123	1 2 769	1 3 098

<sup>1</sup> See 'Methods of preparations' and 'Macroscopical findings'

TABLE 1

*Weight of brain and spinal cord*

	K137 (A)	K107 (N)	K153 (A)	B173 (N)	K1207 (A)	K109 (N)	K172 (A)	AVERAGE NORMAL	AVERAGE AFFECTED
Sex	♂	♂	♀	♂	♀	♂	♂		
Age (days)	300	228	246	424	220	232	230	293	248
Body weight (grams)	343	371	315	358	318	364	339	364	329
Weight of whole brain	1 833	2 004	1 699	1 987	1 686	1 941	1 840	1 977	1 763
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## MICROSCOPICAL FINDINGS

1 *Spinal cord*

The spinal cord of the pigeon has two enlargements, the upper and lower intumescencia. The upper enlargement is located far posteriorly, owing to the bird's long neck, and hence there is a very short thoracic cord between the upper and lower enlargements (cervical vertebrae, 14, thoracic 4, lumbosacral 7 or 8, coccygeal 5). The upper enlargement has a larger diameter than any other part of the cord. At the lower enlargement, the cord is divided into two halves by the 'sinus rhomboidalis,' as named by Kolliker ('02). The two halves of the cord are connected at the lower part of the intumescencia only by the anterior white commissure. According to Kolliker, this sinus is formed by extensive development of the sulcus dorsalis medialis in which there is a gelatinous glial tissue. The ligamentum denticulatum, a band of connective tissue which supports the cord from the lateral edges of the vertebral bodies, appears at the level of the lower enlargement well developed in the anterolateral portion of the cord.

*White matter.* All the sections of the spinal cords at the different levels in the four affected pigeons are decidedly small in reference to both the white and gray matter as seen with the microscope as well from the exact measurements, compared with the sections from the corresponding levels of the normal control birds. The myelin sheaths stained by the Pal-Weigert are generally slightly paler, so that each section of the affected specimens looks as if it were cut much thinner than the normal section, whereas, in fact, they are both exactly the same in thickness, as already indicated. Nevertheless, there is not found any area in the funiculi totally without color by Pal's method.

Throughout all levels of the spinal cord there is a relatively pale area in the median portion of the anterior funiculus and in the dorsolateral periphery of the lateral funiculus, while on the other hand the whole dorsal funiculus is pale. The other portions of the different funiculi do not exhibit any marked color change.

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TABLE 3<sup>1</sup>  
*The upper cervical region of the spinal cord*

	K207 (A)	K199 (N)	K137 (A)	K167 (N)	K158 (A)	B473 (N)	K172 (A)	AVER AGE NOR MAL	AVER AGE AFFEC TED
Transverse diameter of cord in millimeters	2 738	3 340	2 137	2 204	1 970	3 206	1 870	2 595	2 097
Ventrodorsal diameter	1 903	2 171	1 753	1 887	1 118	2 004	1 720	2 020	1 745
Greatest breadth of ventral horn	0 217	0 283	0 250	0 350	0 317	0 334	0 217	0 322	0 250
Distance from the central canal to the latero-anterior periphery of the ventral horn	0 417	0 417	0 450	0 534	0 467	0 534	0 450	0 495	0 442
Greatest breadth of the dor- sal horn	0 133	0 183	0 233	0 301	0 233	0 300	0 150	0 272	0 188
Distance from the dorsal periphery of the dorsal horn to its base	0 300	0 300	0 384	0 417	0 417	0 434	0 384	0 384	0 370
Greatest breadth of the funiculus ventralis	0 417	0 450	0 450	0 534	0 501	0 584	0 501	0 522	0 467
Greatest breadth of the funiculus dorsalis	0 250	0 350	0 384	0 417	0 334	0 417	0 300	0 395	0 317
1) Number, and 2) size of large ganglion cells in the anterior portion of the ventral horn ( $\mu$ )	1 8 2 19.9	16 37.1	3.5 25.7	8.5 28.5	4.5 28.5	11 42.7	6 28.5	11.8 36.2	5.5 25.6
Caliber of the fibers at the medial portion of the funic- ulus anterior ( $\mu$ )	7.9	11.9	8.5	11.4	8.5	10.2	7.2	11.1	6.1
Caliber of the fibers at the dorsolateral portion of the funiculus lateralis ( $\mu$ )	5.7	8.5	2.2	7.1	4.2	6.8	5.5	7.5	4.4
Caliber of the fibers of the funiculus dorsalis	1 1.9 2 2.8	2.8 3.5	1.7 2.2	2.6 2.8	1.9 2.8	2.7 3.4	2.4 3.1	2.8 3.2	2.0 2.7
( $\mu$ ) 1) medial portion, 2) Lateral portion									

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here are small in caliber, yet the difference between the affected and normal is well defined. The lateral area of the funiculus has a slightly larger caliber of fibers than the median portion, but even so they are smaller than those in the normal. In the upper enlargement, the medial triangular area in this funiculus along the median sulcus stains a deep blue to black in the normal, while in the affected one it shows a paler color and reduced breadth. The measurements of fibers are given in table 3. In the lower enlargement, the funiculi posteriores are not attached to each other, but they are separated by a wide space, the sinus rhomboidalis, the sinus side of each funiculus is convex, while the other side is closely applied to the dorsal horn. The funiculi in the affected specimens are not reduced ventrodorsally, but do show a diminution transversely. There appear to be two kinds of fibers in the funiculus in the normal pigeon. One occupies the medial fourth and measures  $3.1\ \mu$  on the average, while the other group occupies the lateral three-fourths and has fibers of much larger caliber,  $5.1\ \mu$ . In these two areas of fiber groups, in the affected specimens, the fibers have a smaller caliber and thinner myelin sheaths, the average caliber of the fibers in the medial portion is  $2.2\ \mu$ , while those in lateral portion measure  $4.2\ \mu$ . Moreover, the arrangement of the fibers is looser.

No segmentation or decoloration in the myelin sheaths of the nerve fibers is seen.

*Gray matter.* The gray matter of the spinal cord of the affected birds shows a reduction in both the anterior and posterior horns and in the central gray matter. Both horns are reduced especially in width, as shown in the tables (3 and 4), but there is not much reduction in length. There is, then, only a decided meagerness of both horns. Besides these changes, other conditions may be pointed out.

In the anterolateral portion of the ventral horn, the ganglion cells are only half the normal in number and the large cells present in the upper and lower enlargements are decidedly reduced in size (table 3), and usually have a slender shape, but are seldom shrunk. In both enlargements, a small area of ganglion cells at the medio-anterior portion of the ventral horn protrudes into

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of fibers around the cells is not pronounced, though a few tiny bundles enter from the dorsal roots and from the funiculus posterior. Schacherl ('02) says the small size of this column in the pigeon makes it difficult to study the internal structure of the individual cells, and he says that the cell group in the upper cervical cord which corresponds to Clarke's column cannot be distinguished.

In the upper enlargement, cervical intumescencia, is found an analogous structure to Clarke's column in full development, but this is different from the arrangement in man and other mammals, for in these it does not appear at this level. It has a large spherical form on each side at the base of the dorsal horn, having its larger diameter from the medioventral to the laterodorsal side and its shorter diameter from the mediodorsal to lateroventral side. Its huge structure, 0.334 to 0.384 mm in diameter in the average normal pigeon, occupies almost the whole space of the gray matter at the base of the dorsal horn. The marked protrusion into the white matter of the posterior funiculus as seen in man does not occur in the pigeon, the whole group of cells is within the horn or in the central gray matter and causes no bulging.

The cells are generally round, oval, sometimes polygonal, they vary in number from ten to eighteen, and among them there are three to six large cells in each average section. The size of these large cells in the normal varies from 31.3 to 51.3  $\mu$  in diameter. The area of this cell group is filled and surrounded by a mass of fine network of fibers which is mainly composed of fibers from the posterior roots and fiber bundles from the funiculus dorsalis. This network of fibers gives the dorsal and medial borders of the column a clear definition from the surrounding tissues. The ventral and lateral borders, however, are not quite so well defined, owing to the diffuse transition into the lateral proprial fasciculus or into the fiber network of the lateral portion of the central gray matter.

In the thoracic region the column decreases in size, the contained cells are less numerous and the network not so luxuriant, but in the lower enlargement, intumescencia lumbosacralis, it

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No round-cell infiltration is seen surrounding the central canal. Sometimes there seems to be a slight increase of neuroglia at the periphery along the medial sulcus in the funiculus posterior in a few sections, however, this is not seen in successive series of preparations. Lissauer's zone and both spinal roots in all the affected birds show no evident difference from the normal.

## 2 *Cerebellum*

The cerebellum of the pigeon lies as a dorsal cover of the fourth ventricle and anteriorly it is attached closely to the large optic lobes. It has a spherical shape and is supported on both sides by a stalk, the crus cerebelli ad medullam (Stieda, '69). This is the only cerebellar peduncle the pigeon has, any independent structure corresponding to the other two peduncles in mammals is not defined, at least macroscopically. The lamellae of the cerebellum, which run transversely on the surface, converge in the crus cerebelli, fan-shaped at the lateral part of the cerebellum. The lateral hemisphere is not present in the pigeon. As to the division of the vermis, Shimazono ('12) divided it into two parts, the vermis anterior and posterior, Ingvar ('18) lately divided it into the lobus anterior, medius, and posterior. The anterior, Ingvar divides from the medius by fissure primarius and the posterior from the medius by the fissure prepyramidalis. He came to this division as a result of his phylogenetic and ontogenic studies. The lobus anterior is divided into four lobuli, lingula, lobus centralis, and culmen, and the lobus posterior into three lobuli, uvula, nodulus, and pyramis. The interplaced lobuli between the anterior and posterior comprise the lobus medius which consists of three lobuli.

A small appendix from the posterolateral portion of the cerebellum turns up, making a furrow between it and the cerebellar body, it is shaped like an auricle, and is called the lobus lateralis or auricle. Many authors agree that it corresponds to the floccular body in mammals. The auricle consists of two main lamellae, the one large anterior, the paraflocculus, the other

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### 3 Brain stem

The medulla oblongata of each of the pigeons, both affected and normal, with the attached half of the cerebellum was studied in the successive serial sections from the distal end up to the height of the oculomotor nucleus in the midbrain. Since the normal structural relations of nuclei and tracts are not very well known, I have described, of necessity, the normal structure first before attempting to point out any alteration or difference in the affected birds.

*The distal portion of the medulla oblongata* At this level of the medulla oblongata, the gray substance of the posterior horn increases in its horizontal width, the axis of the horn is directed laterally, and it contains many myelinated fibers. The myelinated fibers in the posterior horn converge ventromedially at the neck of the horn, whence they run either into the funiculus lateralis, or the anterior horn, while more proximally they also enter the anterior commissure.

The anterior horn is narrower than in the cervical cord, its axis nears the midline, and it contains an abundance of fibers which go to form a part of the thick anterior commissure. The anterior commissure also receives fibers from the lateral funiculus and from the posterior horn. These fibers in the anterior commissure disappear in the opposite medial edge of the funiculus anterior. Going more proximally, the anterior horn becomes smaller and there appears the nucleus hypoglossus, while laterodorsally to the central canal the beginning of the vagus nucleus is seen.

The fibers in the anterior funiculus correspond to the fasciculus longitudinalis medialis of mammals. This has been established by the early formation of myelin sheaths and the pathway taken by these fibers to the midbrain (Brandis, '93). Many fibers in the funiculus anterior and lateralis, which cross in the raphé and run laterally and dorsally, correspond to the internal arcuate fibers of mammals. The thick fiber bundle which runs along the ventral periphery of the medulla forms the external arcuate fibers and makes one of the important connections with the cerebellum.

### 3 Brain stem

The medulla oblongata of each of the pigeons, both affected and normal, with the attached half of the cerebellum was studied in the successive serial sections from the distal end up to the height of the oculomotor nucleus in the midbrain. Since the normal structural relations of nuclei and tracts are not very well known, I have described, of necessity, the normal structure first before attempting to point out any alteration or difference in the affected birds.

*The distal portion of the medulla oblongata* At this level of the medulla oblongata, the gray substance of the posterior horn increases in its horizontal width, the axis of the horn is directed laterally, and it contains many myelinated fibers. The myelinated fibers in the posterior horn converge ventromedially at the neck of the horn, whence they run either into the funiculus lateralis, or the anterior horn, while more proximally they also enter the anterior commissure.

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(Brandis, '93 a, Winkler, '91, Wallenberg, '03) This area of fiber tracts is not so well defined in regard to the separate fibers as in the normal, owing to the poor development of the myelin sheaths and small size of the fibers. This is one of the striking differences in all affected birds from the normal. The internal arcuate fibers which appear more proximally in the medulla oblongata are less distinct than those of the normal, this is especially easy to see at the raphe, where they decussate. The thickness of the external ventral arcuate bundle, which runs ventrally along the periphery of the medulla, gathering fibers from the posterior and also lateral funiculus, is reduced by one-third of the normal, the individual fibers are much thinner in reference to the myelin sheaths and are also reduced in number. This finding makes another striking difference in all affected birds not only at this level, but also at other levels of the medulla.

*The level of the nucleus olivaris inferior* The nucleus olivaris inferior in the pigeon appears at the level of the hypoglossal root and lateral to it, but it is partly pierced by this nerve in the ventral region of the medulla oblongata. It lies in a transverse position, having a thick gray mass at the lateral end. Brandis only described "ein grosses fast faserfreies Feld" at this level without paying any further attention to the olivary nucleus. Yoshimura ('10) found experimentally that by injury to the cerebellum this nucleus degenerated almost totally contralaterally. The olivary nucleus is connected by way of the arcuate fibers to the cerebellum homo- and contralaterally and has a direct connection with the spinal cord. The median part of this nucleus contains a rich fiber network with few cells. Shimazono infers from his experimental and embryological study that almost certainly fibers from the olivary nucleus cross in the raphe and go dorsally to the cerebellum.

In all our preparations except only the case of the pigeon no K207, the nucleus olivaris inferior is poorly developed, the nucleus is small, the normal measures transversely 0.668 mm and ventrodorsally 0.367 mm, while the affected specimens measure 0.418 mm transversely and 0.117 mm ventrodorsally.

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The zone of *fibrae arcuatae externae ventrales* is extremely thin and contains only a few fibers and stains less intensely than normal. The area of the *fasciculus long med* and *pre-dorsalis* shows diminished transverse as well as ventrodorsal dimension, especially the former. In the distal portion of the medulla oblongata, this *fasciculus long med* cannot be separated from the fiber tract which lies ventrally to this *fasciculus*. The area of this *fasciculus* contains many smaller fibers, all more variable in size than the normal, the average caliber is  $5.7 \mu$  in the ataxic and  $8.6 \mu$  in the normal, the staining also not so deep as in the normal. Nucleus of the *ala cinerea* and the fibers of the *vaguglossopharyngeus* are normal.

All above findings are quite uniform in all affected pigeons, there being only a difference in degree.

*The level of the cochlear nuclei.* The acoustic nerve enters the medulla oblongata in two roots as in mammals, the one is the dorsal, distal, or lateral root, the *nervus cochlearis*, the other is the ventral, proximal, or medial root, the *nervus vestibularis*. The nuclei of the acoustic nerve are divided into three main groups. One, the nucleus angularis, 'Eckkeirn,' located at the dorsolateral portion of the medulla, into which the cochlear root enters, this corresponds to the *tuberculum acusticum* of mammals (Brandis, '94, Winkler, '91). The second nucleus appears in the lateral wall of the fourth ventricle, crescent-shaped, surrounded by a medullated fiber mass, and is named the nucleus parvo-cellularis, 'der kleinzellige Kern'. The third one, the largest, occupies the space between the above two nuclei, nucleus magno-cellularis, 'der grosszellige Kern'. The magno-cellular nucleus is supposed to be analogous to the nucleus Deiters in mammals.

The nucleus angularis as well as the cochlear stem in all four ataxic birds are almost the same in the normal in reference to their developmental conditions. The nucleus parvo-cellularis has sometimes a little narrower shape and the medullated fibers around it appear to be slightly reduced. The nucleus magno-cellularis, though it appears almost the same as normal, seems to be diminished at its proximal dorsal portion in the

The zone of *fibrae arcuatae externae ventrales* is extremely thin and contains only a few fibers and stains less intensely than normal. The area of the fasciculus long med and predorsalis shows diminished transverse as well as ventrodorsal dimension, especially the former. In the distal portion of the medulla oblongata, this fasciculus long med cannot be separated from the fiber tract which lies ventrally to this fasciculus. The area of this fasciculus contains many smaller fibers, all more variable in size than the normal, the average caliber is  $5.7 \mu$  in the ataxic and  $8.6 \mu$  in the normal, the staining also not so deep as in the normal. Nucleus of the ala cinerea and the fibers of the vagoglossopharyngeus are normal.

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the spinocerebellar tract and bundles from the arcuate fibers which form the main portion of the restiform body, and is observed in all serial sections in all the affected birds

The fiber bundle along the ventral periphery of the medulla is quite thin in all affected pigeons. This is indeed very striking, the ventrodorsal thickness of this bundle measures 0.100 mm in the normal and 0.043 mm in the affected bird—a dimension of less than one-half the normal. There is not only a reduction of thickness of the bundle, but each individual fiber is smaller and stains weakly owing to its thin myelin sheaths. The above condition of this bundle throughout its course is common in all affected birds and it constitutes one of the most decided changes in the medulla.

The internal arcuate fibers are less prominent and quite indistinct, owing to the reduction in fibers, their poor staining properties and the small size of the individual fibers, the scattered large ganglion cells between the fibers in the reticular formation are not only few in number, but also small in size, the average size of the large cells being 22.8 to 37.5  $\mu$  in the affected and 28.5 to 57.0  $\mu$  in the normal. The area of the reticular formation is apparently reduced.

The tall triangular area of the longitudinal fiber bundle along the raphé with its base directed dorsally to the floor of the fourth ventricle, that is, the area of the fasc. long. med. and predorsalis is smaller, especially in its transverse diameter, this measuring 0.638 mm in the affected and 0.985 mm in the normal. The fibers which cross the raphé from side to side and the fibers which run ventrodorsally in the raphé in this area are quite scant. The fibers, both the longitudinal and transversal, are thin, indistinct, and do not stain a deep black by Weigert. The spinal root of the trigeminal nerve shows a good development in both the healthy and unhealthy birds.

*The level of the vestibular nerve.* Proximal and a little ventral to the cochlear stem, the nervus vestibularis appears as a large bundle entering the medulla oblongata. A part of the fibers runs dorsally to the acoustic area, to the nucleus magno-cellularis, while the other and greater part of the fibers runs medially

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The fiber groups which go dorsally and to the ventral portion of the acoustic area and which pass into the ventral part of the nucleus parvo-cellularis are slightly smaller than normal. In the ventral region of the cerebellar peduncle, between the medial edge of the spinocerebellar tract and the lateral region of the 'Bogenzug,' we find an area of large ganglion cells which is the proximal process of the nucleus magno-cellularis. The transverse breadth of this nucleus here measures 0.835 mm in the affected and 1.084 mm in the normal, its dorsoventral boundary is somewhat difficult to make out because of the close relation to the ventral cells of the nucleus cerebellaris lateralis. The cerebellar peduncle has a small breadth and the lateral fiber mass in this peduncle which is composed of fibers that come mainly from the medulla oblongata has a breadth of 0.334 mm in the affected and 0.835 mm in the normal, or a ratio, therefore, of 1:2.5.

The difference of the external ventral arcuate fibers between the normal and affected becomes more prominent than ever at this point, 0.100 mm in normal and 0.065 mm in the affected birds. The staining properties, caliber, and other conditions of each individual fiber are the same as described before. It is clearly seen that the *fibrae arcuatae externae ventrales* run latero-dorsally and enter the restiform body and with the *tractus spinocerebellaris* pass dorsally up to the lateral edge of the cerebellar peduncle. The fibers in the reticular formation have the same changes as noted at the former level. The changes in the *fasciculus longitudinalis medialis*, the external ventral arcuate fibers, the lateral portion in the cerebellar process, and the restiform body are the main important differences in the affected pigeons at this level with, however, no apparent degenerative processes in either fibers or nuclei.

The facial nerve and its nucleus appear normal.

*The level of the sensory trigeminal nucleus.* The spinal root of the trigeminal nerve is pierced ventrodorsally by a few fibers which come from the reticular formation and pass toward the

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parently diminished in number. This nucleus appears in frontal section, and proximally disappears at the level where the trochlear nucleus shows its largest size. In the affected preparation, on the other hand, the nucleus disappears at the level just proximal to the place of appearance of the sensory nucleus of the trigeminal nerve.

This nucleus has never been exactly described, even though it seems to be similar to the oliva in Friedlander's illustration ('98) or the medial  $\alpha$ -nucleus ('Trapezkern,' Westphal) of Wallenberg ('04) in reference to its localization. The inferior olivary nucleus of the pigeon lies, however, far distal and lateral to the hypoglossal root, while the superior olivary nucleus lies more dorsalward, as already described. If this nucleus had a connection with the fiber bundle of the ventral system of the eighth nerve, like the superior olivary nucleus and the nucleus isthmi, it would be normal as these are. Thomas ('11) reports the atrophy of the arciform nucleus in cerebellar atrophy, and contends from his experiments that the arciform nucleus has close relation with the pontine nucleus. It seems to me rather, therefore, that this nucleus may have some close physiological relation to the already described ventral fiber bundle or to the arcuate fibers than to the ventral system of the eighth nerve.

The internal arcuate fibers in the reticular formation are fewer and paler, differences especially marked in the fibers which cross at the raphé. At the lateral edge of the brain stem, a fiber mass runs toward the cerebellum passing through the cerebellar peduncle, which measures 0.25 mm. in the normal and 0.08 mm. in the affected birds. Going more proximally from this level, the fasc. long. med. becomes more and more round in section and the cerebellar peduncle decreases in breadth.

At this point it will be well to describe more thoroughly the cerebellar peduncle, from its distal to proximal end, for reduction of this peduncle is one of important differences in the affected birds.

The distal connection of the medulla oblongata to the cerebellum in frontal sections begins at the level of appearance of the cochlear nerve. The spinocerebellar tract at the lateral

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nucleus of the trigeminal nerve as a fiber mass running from the ventral part of the cerebellar peduncle to the medial part of the medulla. This brachium is perfectly normal in the affected specimens. The decussation of the brachium as well as the nucleus ruber at the proximal region of the oculomotor nerve are well developed.

As to the connection of the cerebellum to the fasciculus longitudinalis medialis, no definite structure could be identified.

*The levels of the trochlear and oculomotor nuclei.* The trochlear nucleus, its size, shape, and number of cells, together with the fibers coming from the nucleus in the affected specimens, all appear as in the normal sections. The whole dorsoventral diameter of the brain stem at this level of the nucleus trochlearis measures in the midline 3.507 mm in the normal and 2.922 mm in the affected section. The entire width of the brain stem from side to side measures 5.511 mm in the normal and 4.843 mm in the affected. These measurements show a reduction of the brain stem at this level, but the small size is chiefly due to a diminution in area of the reticular formation. The ventral arcuate bundle has already disappeared.

The oculomotor nucleus appears proximal to the trochlear nucleus as its continuation, medial and slightly dorsal to the fasciculus longitudinalis medialis. The dorsolateral centers of this nucleus contain small well-defined cells, but the other centers have large cells. From the nucleus fibers emerge passing ventrally, part of them crossing the raphé, where they form the internal fibers of the opposite oculomotor nerve. At the point of emergence of the nerve fibers from the brain stem, there appears in the nerve stem a half-moon-shaped, colorless line in the Weigert preparation. This condition may be a structure analogous to that already discussed in the cranial nerve roots in the human by Thomsen ('87), Hülles ('06) and Staderini ('90). No pathological or defective development in the oculomotor nerve fibers or in its nucleus is visible in the affected pigeons.

The fasciculus longitudinalis medialis in this region is small and appears less oval than the normal, but the reduction is not as marked as at the former levels, it measures transversely 0.317 mm in the affected,

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marked in the anterior portion. The fibers from the reticular formation to the peduncle are hardly recognizable.

In the medulla oblongata, the most conspicuous and constant changes in all affected birds are the reduction of the fiber bundles in the ventral periphery of the brain stem, the fasciculus longitudinalis medialis, the cerebellar peduncle, and the reticular formation, including the internal arcuate fibers and ganglion cells. Other differences, though not so considerable as in the above structures, and sometimes not constant, may be pointed out: the reduction of the nuclei funiculi posteriores, nucleus olivaris inferior with its inter- and circumolivary fibers, and a symmetrical nucleus which is located at the ventral periphery in the middle portion of the brain stem. All the cranial nerves are normal in nuclei and fibers in all affected birds. The nucleus ruber, the brachium conjunctivum, the nucleus isthmi (nucleus lemniscus lateralis) in the midbrain, and the nucleus mesencephalicus lateralis in the optic lobe appear in good condition.

In addition to the small size of the gray and white matter in the spinal cord en masse, there is decidedly poor development of cells in Clarke's column, the anterior horn, and the central gray matter, all those present being small in size, often one-half the normal, few in number, with scanty processes. The median portion of the funiculus anterior, the direct spinocerebellar tract, and the posterior funiculi are reduced in area and also show indistinct borders owing to the intermingling of abnormally small and delicate fibers which show a diminution of caliber. The fiber network in all the gray matter, especially, in Clarke's column is scant. The capillaries and small blood-vessels in the substance of the spinal cord and cerebellar cortex are apparently greatly reduced. None of the spinal roots or Lassauer's zone show any variations. In all cases there is never seen a definite degenerative or regressive process, such as segmentation of myelin sheaths, or increases of the neuroglia or interstitial tissues. No thickening of the pia mater, the coats of the blood-vessels, or abnormal cell infiltration is observed.

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Under the name of hereditary ataxia, we understand a condition of congenital disturbance of coordination of movements or even in standing, static ataxia. This disease is generally classified into two types: one is the Friedreich's ataxia and the other is the L'hérédo-ataxia cérébelleuse. The former was first described by Friedreich in 1863, who noticed a typical hereditary form of a chronic degenerative atrophy in the posterior and lateral funiculi of the spinal cord. The affection is characterized clinically by a disturbance in the coordination of movements, as he says "Die Krankheiten dürfte vom klinischen Gesichtspunkte aus als chronische progressive Lahmung der Combination der Bewegungen, von pathologisch anatomischen Standpunkte aus als chronische degenerative Atrophie der spinalen Hinterstränge zu bezeichnen sein." After his discovery of this affection, similar cases were reported by Carpenter ('71) and Gowers ('80) in England, and Brousse in France ('82), who had proposed to call the affection by the name of Friedreich's disease (Marie, '92). The chief changes in this disease are usually a thin small cord, with degeneration or atrophy, and, consequently secondary sclerosis of the lateral and posterior columns, and thickening of the pia mater. The frequently affected tracts are in the proprioceptive systems in the cord, namely the direct spinocerebellar tracts and the columns of Goll and Burdach, but sometimes the lateral pyramidal tract is also involved. Occasionally there is more or less diminution in the number of fibers in the anterolateral column, and also atrophy of the cells in both the anterior and posterior horns (Blocq and Marinesco, '90, Barker, '03). The cells of the column of Clarke are notably degenerated or else very poor in development. All authors agree in that this disease is due to an arrest of development or growth of the various systems of fibers in the spinal cord (Menzel, '91).

Under the title of L'hérédo-ataxia cérébelleuse, Marie ('93) reported two cases of a familial ataxia, and collected from the literature a series of similar cases, in which atrophy of the cerebellum was found. He has established a symptom-complex distinct from Friedreich's ataxia. Marie's disease is believed to be

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As to the real etiology of this disease, it has been assumed that many factors may play a part, such as idiocy, epilepsy, alcoholism, acute infectious diseases, and consanguinity of the parents (Oppenheim, '00, Starr, '09), but none of these have much real value. The connection cannot be made. They lack positive proof as etiologic factors.

As circumstantial pedigrees show, all our cases descend from an original egg (pigeon female, no. 151) produced by the weakening influence on a reproductively overworked normal parent (from records of Dr. Oscar Riddle). Whether or how this overwork has a direct influence on the central nervous system or exerts some secondary effect on this system as a result of some nutritional changes is a further difficult problem. Nevertheless, we feel that we can deduce the very interesting fact that some disorder or disturbance caused by 'reproductive overwork' was at least an important etiological factor of this disease, and that the basis for such a conclusion in the present case does not rest on uncertain observations, as has often been done previously, but upon a practical and experimental foundation.

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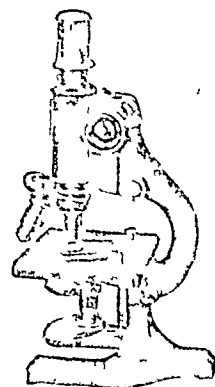
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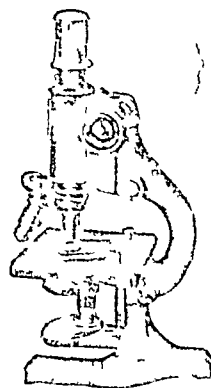
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Resumen por el autor, A. C. Ivy  
Universidad de Chicago

## Estudios experimentales sobre el tallo cerebral

### II Un estudio comparativo de la relación de la corteza cerebral con el nistagmo vestibular

El autor ha estudiado la relación de la corteza cerebral con el nistagmo vestibular en la rana, tortuga, paloma, conejo y gatos y perros jóvenes y adultos. Llevó a cabo varias ablaciones del cerebro observando su efecto sobre el nistagmo vestibular, usando como estímulo la rotación. La extracción del cerebro en la rana, tortuga y paloma no perturba el nistagmo vestibular. La extracción completa del cerebro en el conejo con la destrucción extensa del tálamo no suprime el componente rápido del nistagmo, siempre que la temperatura del cuerpo se conserve normal. Las observaciones de F. T. Rogers sobre la reducción de la temperatura del cuerpo subsiguiente a las lesiones del tálamo y su efecto sobre los reflejos ha sido confirmada por el autor en el conejo. En el gato y perro la ablación de la corteza motriz en la región del área ocular causa un aumento temporal, con alguna permanencia, de 5 a 15 veces mayor, en la duración de post-nistagmo, cuando se hace girar al animal sobre el lado de la lesión. Hay un aumento en la reacción del nistagmo cuando la desviación es opuesta al lado de la lesión, con alguna disminución, pero no cesación cuando la desviación tiene lugar hacia el lado de la lesión. Los hechos apuntados dan validez a la conclusión de que el componente rápido del nistagmo vestibular no se debe a la integridad de un arco reflejo cerebral, sino que depende de algún centro situado debajo del tálamo, sobre el cual ejerce el cerebro una bien reconocida acción inhibitoria.

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Observations were made upon rotatory and postrotatory nystagmus. The animals were placed upon a turntable and rotated, the speed and number of rotations being controlled.

### RESULTS

*Frog* Some observers have reported the presence of compensatory movements of the eyes of the frog, others have not. This discrepancy is probably due to the condition of the frog and the acuteness of observation. Out of about one hundred frogs examined only a few were found that did not show on rotation a true vestibular nystagmus with slow and quick components. The small green frog (*Rana pipiens*) and the jumbo bull-frog (*Rana catesbeiana*) were used. The latter is much better than the former for study, as the nystagmus reaction is more marked and the eyes are larger. Several precautions are necessary in order to observe the reaction. It is necessary to rotate the frog slowly; if rotated too rapidly, only deviation occurs. The quick component is very slight in degree ( $\frac{1}{8}$  to  $\frac{1}{32}$  inch, depending on the size of the frog), for the deviation in the frog is not marked. It is easier to observe, if the head is held between the fingers to prevent head nystagmus. If the frog struggles much, it will be absent. Pinning to the frog board often inhibits the reaction. Postrotatory nystagmus is very infrequent. I have observed it, however, consisting only of one or two movements. The temperature of the frog is very important. The best reaction occurs at 18° to 20°C.

Decerebration in the frog never abolishes the quick component of nystagmus nor interferes with the nystagmus reaction in any way. If the frog is depressed as a result of the operation, then deviation only is observed.

*Turtle* Wilson and Pike ('15) report that nystagmus is absent in the turtle. My observations are to the contrary, provided the turtle's temperature is between 10° and 39°C. On either side of these temperatures the quick component is abolished and deviation only is present.

This effect of temperature upon nystagmus is only to be expected when it is recalled that reflexes in general are depressed by temperature on either side of the normal or optimum.

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is present in the pigeon only to a slight and less pronounced degree than in mammals. According to my observations, the average number of rotatory nystagmic movements for thirty pigeons<sup>1</sup> when rotated through an arc of ninety degrees at a speed of one turn in two seconds was six, the minimum number being three, the maximum ten. The average number of postrotatory movements for the same group of pigeons when rotated ten times at a speed of one turn in two seconds was eleven, the minimum being four, the maximum twenty. The average duration of the after-nystagmus was five seconds.

Hemi-decerebration in the pigeon has no effect upon nystagmus.

Complete decerebration with even extensive injury to the thalamus does not abolish the quick component of nystagmus provided the temperature of the bird is kept normal. Rogers (18) has shown that the temperature of the decerebrate bird with thalamic lesion must be kept normal in order to get normal decerebrate behavior. In two such pigeons, whose body temperature fluctuated with the temperature of the surrounding air, it was found that the quick component of nystagmus disappeared at 34°C in one and 35°C in the other, while deviation persisted.

*Rabbits* True vestibular nystagmus is present in the rabbit. The average number of rotatory movements for eight rabbits when rotated through an arc of ninety degrees at a speed of one turn in two seconds was five, the minimum being four, the maximum seven. The average number of postrotatory movements for the same group of rabbits when rotated at the same speed was sixteen, the minimum being seven, the maximum twenty-four. The average duration of the after-nystagmus was eight seconds.

Some rabbits show a marked variation in the number of movements and the duration of the after-nystagmus, although they were rotated at the same rate of speed and other factors were controlled. One of the rabbits varied from seven to twenty-two movements, or from four to ten seconds, during the course of

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Complete decerebration in the rabbit does not abolish the quick component of nystagmus. The entire thalamus can also be destroyed (figs 8 and 9) without abolishing the quick component. In the decerebrate rabbit the quick component persists until the animal becomes depressed because of degenerations involving lower centers or because of inanition, it being very difficult to keep these animals in a good state of nutrition. In the decerebrate rabbit with destruction of the thalamus the temperature becomes subnormal and the quick component disappears, but will return again if the animal is placed in an incubator and its body temperature raised to normal. Deviation still persists with subnormal temperature. In such a rabbit immediately after the operation and for four to five hours later the quick component is very manifest, but after this time it is irregular and subject to wide variations. Two such animals manifested no rotatory quick component when tied to a board, but when held in the hands and rotated the quick component was present. Without taking into consideration these last two points, along with body temperature, one might overlook the presence of the quick component in rabbits without cerebrum and thalamus.

The rabbit is a convenient animal in which to demonstrate the presence or absence of the quick component following various brain lesions.

*Cats.* Six cats have been worked upon with the same results as observed in the rabbit and the dog.

*Kittens and pups.* The same observations hold true in young animals as observed in the adult, except for a general rule that the depression from the operation is less marked and the effects produced are more temporary. However, one pup, which was operated at the age of four months and is now one year old (August 1, 1919), in which the left motor cortex, occipital cortex and basal portion of the temporal lobe was extirpated, now shows three postrotatory movements when rotated to the left, and eight when rotated to the right. The increase in the after-nystagmus when rotated opposite to the side of the lesion seems to be permanent in this pup.

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normal ( $32^{\circ}$  to  $35^{\circ}$  C.) and it was very depressed, being comatose forty-eight hours previous to death. The quick component was absent during the last forty-eight hours. Autopsy revealed the thalamus to have undergone complete malacia. At the time observations were being made on this dog I was not aware of the effect of body temperature upon the quick component of nystagmus and hence did not study the effect of raising the body temperature as was done in the case of the rabbits reported above.

A clean ablation of the occipital cortex in the dog does not alter vestibular nystagmus as judged by the results from such a procedure in three dogs.

#### DISCUSSION

Tozer and Sherrington ('10) have demonstrated histologically and physiologically the presence of sensory tendon nerves in the extrinsic eye muscles which pass back to the midbrain via the IIIrd, IVth, and Vth nerves. Wilson and Pike ('15) suggest that afferent impulses from these tendon nerves "set up efferent impulses in the oculomotor cells of the cerebrum, which result in a quick jerky contraction of the internal rectus on the side of the slow deviation and of the external rectus of the opposite side, with relaxation of the antagonistic muscles," which effects a restoration of the eyes to the primary position. In other words, these latter investigators are of the opinion that the quick component is dependent upon the presence of the neopallium or upon the integrity of a cerebral reflex arc. The presence of true vestibular nystagmus in the frog, pigeon, and turtle questions this idea from the viewpoint of comparative anatomy. The persistence of true vestibular nystagmus following the removal of the cerebral hemispheres in these forms) which is a very simple matter and causes no great physiological disturbance—questions this idea physiologically. The observation that no disturbance of nystagmus follows decerebration in these forms, while in the higher forms (rabbit, cat, dog) there is a change in the number and duration of the nystagmic movements, shows that some constituent is absent in the cerebrum of the frog, turtle, and pigeon which is present in the cerebrum of the rabbit, cat, and dog.

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## PLATE 1

### EXPLANATION OF FIGURES

1 Dog 18 Ablation of the posterior two-thirds of the left cerebral hemisphere including the basal portion of the temporal lobe and motor cortex. This dog showed typical increase in nystagmus when slow component was directed opposite to the side of the lesion. Quick component was present when rotated in either direction.

2 Dog 31 Complete left hemi-decerebration, including destruction of the left half of the thalamus. See table 3. Seen from injured side.

3 Dog 31 Ventral view. Both third nerves and midbrain are intact. This dog showed nystagmus when rotated in either direction with an increase as shown in table 3. *a*, Third nerves.

4 Dog 32 Complete left hemi-decerebration, including destruction of left lateral and middle portions of the thalamus and anterior left half of midbrain. Nystagmus was absent in this dog which lived only three days. *a*, Right third nerve intact. *b*, left third nerve absent with injury to midbrain.

5 Dog 4 Hemi-decerebration. *a*, Thickened dura, *b*, section made through fibrous tissue for examination of thalamus, which showed areas of degeneration.

6 Dog 5 Complete left hemi-decerebration with injury to lateral portion of thalamus done in two operations. Posterior two-thirds of right cerebral hemisphere removed at a third operation. There was an increase in nystagmus when the slow component was opposite to the side of the fresh lesion. The quick component was present when rotated either direction. Dog was killed three days after the third operation by another dog. See table 3. *a*, Temporal muscle drawn inwards through defect in the skull, *b*, thickened dura, *c*, hemorrhagic remaining portion of cerebrum (the only cortex present), *d*, foramen.

7 Rabbit III Left hemi-decerebration with injury to lateral portion of the thalamus. See table 3. *a*, anterior quadrigemina.

8 Rabbit VII Complete decerebration and destruction of the thalamus. *a*, Anterior quadrigemina, *b*, optic nerves and chiasma.

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#### III Los efectos de extensas variaciones de la temperatura del cuerpo causadas por las lesiones del tálamo, sobre las actividades reflejas

La extracción de los hemisferios cerebrales y el tálamo en la palomo reduce al animal a una condición poikiloterma permanente. Uno de estos animales así operado puede conservarse vivo durante un periodo de 1 a 3 meses, colocándole en una incubadora a 30°C. El comportamiento ulterior y las actividades reflejas varían con la temperatura del cuerpo. Los movimientos indecisos típicos de los animales desprovistos de cerebro aparecen cuando el animal está hambriento, si la temperatura del cuerpo es superior a 36°. Si se deja descender dicha temperatura hasta los 30° aparecen perturbaciones en el equilibrio, que se manifiestan primero por la presencia de una flexión tónica de la pata y músculos del pie. A 24° o a menor temperatura el animal no puede mantenerse en pie o volar. Los reflejos oculares, pupilares y el nistagmo desaparecen a unos 30°. Todos ellos reaparecen cuando la temperatura vuelve a ser la normal de 40°. El autor consigna observaciones fisiológicas detalladas sobre un animal después de la ablación de todas las partes del cerebro anteriores a la comisura posterior y al quiasma óptico, seguidas de un estudio microscópico de cortes seriados de las restantes partes del tallo cerebral.

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Vulpian in 1866 noticed that removal of the cerebral hemispheres of the carp led to continuous excessive activity of the fish, always however avoiding obstacles in its path

Ferrier and Steiner found that removal of the hemispheres of the shark gave the same picture as followed loss of the olfactory lobes, that if the thalamus were removed, the animal lay quietly on the floor of the aquarium without movement. Bethe denied this stating that loss of the thalamus did not abolish spontaneous movements

Steiner, Bethe, and Loeb all agreed that damage to the mid-brain leads to motor disturbances, in the form of forced movements or circus movements, if the lesion is unilateral, in the direction of the intact half of the midbrain

Removal of the hemispheres and thalamus of the frog abolishes spontaneous movements, according to Steiner, but not if the thalamus is left intact (Schrader)

Rolando ('09) studied the effects of decerebration in the pigeon. His observations were confined to birds which lived a few days only after operation

Flourens ('22) continued the work and kept the birds alive for months after operation. He made no distinction between decerebration with and without thalamic involvement

Longet ('47) was the first to attribute significance to sharp localization of brain lesions and prepared decerebrate birds with and without damage to the underlying parts

Munk in 1883, revived the controversy between Flourens, who maintained that the hemispheres were necessary for the various senses and Cuvier, who thought that loss of the forebrain led merely to the loss of memory images

Schrader, in 1889, in an elaborate monograph reported results on decerebrate pigeons in which the thalamus was carefully preserved. His primary interest was in the functions of the cerebral hemispheres and not much consideration was given to the thalamus save to make sure that it was present

Vulpian had previously considered the activities of decerebrate animals as due automatically to stimuli "either internal or external" which incited the movements of the animals

Vulpian in 1866 noticed that removal of the cerebral hemispheres of the carp led to continuous excessive activity of the fish, always however avoiding obstacles in its path

Ferrier and Steiner found that removal of the hemispheres of the shark gave the same picture as followed loss of the olfactory lobes, that if the thalamus were removed, the animal lay quietly on the floor of the aquarium without movement. Bethe denied this stating that loss of the thalamus did not abolish spontaneous movements

Steiner, Bethe, and Loeb all agreed that damage to the mid-brain leads to motor disturbances, in the form of forced movements or circus movements, if the lesion is unilateral, in the direction of the intact half of the midbrain

Removal of the hemispheres and thalamus of the frog abolishes spontaneous movements, according to Steiner, but not if the thalamus is left intact (Schrader)

Rolando ('09) studied the effects of decerebration in the pigeon. His observations were confined to birds which lived a few days only after operation

Flourens (22) continued the work and kept the birds alive for months after operation. He made no distinction between decerebration with and without thalamic involvement

Longet ('47) was the first to attribute significance to sharp localization of brain lesions and prepared decerebrate birds with and without damage to the underlying parts

Munk in 1883, revived the controversy between Flourens, who maintained that the hemispheres were necessary for the various senses and Cuvier, who thought that loss of the forebrain led merely to the loss of memory images

Schrader, in 1889, in an elaborate monograph reported results on decerebrate pigeons in which the thalamus was carefully preserved. His primary interest was in the functions of the cerebral hemispheres and not much consideration was given to the thalamus save to make sure that it was present

Vulpian had previously considered the activities of decerebrate animals as due automatically to stimuli "either internal or external" which incited the movements of the animals

The writer has been accustomed to leave a bridge of bone overlying the longitudinal sinus and then cutting through the dura, parallel to the median sulcus and to the occipital pole of the hemisphere. Hemorrhage from the large superficial artery running over the anterior surface of the hemisphere may be controlled with a cautery and the entire hemisphere removed with a blunt probe whose tip has been curved to fit around the posterior end of the hemisphere. This can be removed, the hemorrhage controlled with cotton, and a clear view of the thalamus and the third ventricle obtained. The writer has then destroyed the thalamus either by excision or by the use of a hot cautery. The latter is more satisfactory, in that it controls bleeding as well as destroying the thalamus.

In the second place, it has never been found satisfactory to leave cotton or any packing in the cranial cavity, but to allow the cavity to fill itself with blood and sewing the skin over the cavity. No attempt was made to approximate the cut edges of the dura, because of its delicacy and the fact that traction on the dura may increase the hemorrhage.

After the most careful operative work it is found that only one-fourth to one-third of the animals will live for more than a few days. These early fatalities seem to be due to circulatory disturbances. The percentage of survivals can be increased markedly if the thin medial and occipital cortex is not removed. These parts are so closely related anatomically to the large blood-vessels of the brain stem that their removal is particularly likely to be associated with excessive bleeding.

#### EXPERIMENTAL RESULTS

Complete removal of all forebrain substance anterior to the thalamus gives a preparation which, if the animal lives over the initial shock, conforms to the classic description. Certain features are characteristic of such an animal.

- 1 The bird stands quietly on one or both feet most of the time.

- 2 The feathers are fluffed as in the sleeping condition.

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- 1 The bird stands quietly on one or both feet most of the time.

- 2 The feathers are fluffed as in the sleeping condition.

*Pigeon 126*

*June 21* Bird decerebrated and thalamus cauterized with a hot probe

*June 22* 9 00 A M Temp,  $36^{\circ}\text{C}$  Bird sluggish, stands with difficulty, toes are slightly arched upward so that the bird tends to stand on the claws only (This condition is referred to as 'claw foot' in the laboratory record) Bird put in a warm incubator kept at  $32^{\circ}\text{C}$

10 00 P M Bird is walking around, temperature of bird is  $43^{\circ}\text{C}$

*June 23* Incubator in which bird is kept adjusted to  $30^{\circ}\text{C}$ , and bird stays in incubator until July 1

*June 26* Bird is preening itself Is fed and watered by hand No difficulty in feeding Temperature of bird,  $41^{\circ}\text{C}$

*June 28* Temperature of bird  $38^{\circ}\text{C}$  Rotatory and post-rotatory nystagmus of eyes when bird is rotated Equilibrium normal, feathers fluffed Difficulties in feeding, as bird rejects much of the food put in the mouth By use of much water, however, some is swallowed

*June 30* Bird quiet, no restlessness when starved

*July 1* Bird removed to room temperature ( $24^{\circ}\text{C}$ ) Temperature of bird falls to  $37^{\circ}$  Pupillary reactions to light present but sluggish

Size of pupil, bright light 3 mm diam

Size of pupil, dim light 4.5 mm diam

Change in size of pupil with every blinking movement of the eyelids

Vomiting Respiration, 22 Equilibrium normal Bird perches on my finger

*July 2* 10 00 A M Bird has been in cool place over night Body temperature,  $33.5^{\circ}$  Respiration, 17 Slight tendency to claw foot Slight disturbances in maintaining balance on a perch Bird preens itself Bird put in incubator at  $34^{\circ}$

11 00 A M Bird squatting on floor Temperature of bird  $36^{\circ}$  Preening

Bird has been starved for forty-eight hours, but no restless walking movements occurred

1 00 P M Temperature of bird has risen to  $40^{\circ}$  Typical decerebrate restlessness, bird walking about the cage This does not cease when bird is given excess water Bird is picked up by hand and put down again Walking movements stop momentarily and then resumed

*July 4* 6 00 P M Very hot day Temperature of room  $34^{\circ}$  Bird has body temperature of  $44^{\circ}$  Bird walking around its cage all day Given water and it becomes quiet Feathers fluffed in normal way Pupils widely dilated

10 00 P M Temperature of bird  $39.5^{\circ}$  Temperature of room  $30^{\circ}$  Bird standing quietly asleep on one foot Feathers slightly fluffed

Turn on light in cage suddenly Bird begins walking around Turn off the light and bird becomes quiet Again the light was turned on The bird appears to wake up Turns its head, moves a few steps, hesitates, and then begins walking around the cage Light removed The bird continues walking in semi-darkness and walks against the walls of the cage Repeats this several times

*Pigeon 126*

*June 21* Bird decerebrated and thalamus cauterized with a hot probe

*June 22* 9 00 A M Temp, 36°C Bud sluggish, stands with difficulty, toes are slightly arched upward so that the bird tends to stand on the claws only (This condition is referred to as 'claw foot' in the laboratory record) Bird put in a warm incubator kept at 32°C

10 00 P M Bud is walking around, temperature of bud is 43°C

*June 23* Incubator in which bud is kept adjusted to 30°C, and bird stays in incubator until July 1

*June 26* Bird is preening itself Is fed and watered by hand No difficulty in feeding Temperature of bud, 41°C

*June 28* Temperature of bird 38°C Rotatory and post-rotatory nystagmus of eyes when bird is rotated Equilibrium normal, feathers fluffed Difficulties in feeding, as bud rejects much of the food put in the mouth By use of much water, however, some is swallowed

*June 30* Bud quiet, no restlessness when starved

*July 1* Bud removed to room temperature (24°C) Temperature of bird falls to 37° Pupillary reactions to light present but sluggish

Size of pupil, bright light 3 mm diam

Size of pupil, dim light 4.5 mm diam

Change in size of pupil with every blinking movement of the eyelids

Vomiting Respiration, 22 Equilibrium normal Bud perches on my finger

*July 2* 10 00 A M Bird has been in cool place over night Body temperature, 33.5° Respiration, 17 Slight tendency to claw foot Slight disturbances in maintaining balance on a perch Bud preens itself Bird put in incubator at 34°

11 00 A M Bird squatting on floor Temperature of bud 36° Preening

Bird has been starved for forty-eight hours, but no restless walking movements occurred

1 00 P M Temperature of bud has risen to 40° Typical decerebrate restlessness, bird walking about the cage This does not cease when bud is given excess water Bird is picked up by hand and put down again Walking movements stop momentarily and then resumed

*July 4* 6 00 P M Very hot day Temperature of room 34° Bud has body temperature of 44° Bud walking around its cage all day Given water and it becomes quiet Feathers fluffed in normal way Pupils widely dilated

10 00 P M Temperature of bud 39.5° Temperature of room 30° Bird standing quietly asleep on one foot Feathers slightly fluffed

Turn on light in cage suddenly Bud begins walking around Turn off the light and bird becomes quiet Again the light was turned on The bird appears to wake up Turns its head, moves a few steps, hesitates, and then begins walking around the cage Light removed The bird continues walking in semi-darkness and walks against the walls of the cage Repeats this several times



## PLATE 1

### EXPLANATION OF FIGURES

Figs. 1 2 3, 4 Photographs of a bird with cerebrum and thalamus removed at different body temperatures. Photographs made at two-hour intervals. Body temperature lowered and raised by putting the bird in a cooled or warm incubator.

- 1 Body temperature 33°
- 2 Body temperature 26°
- 3 Body temperature 22°
- 4 Body temperature 39°

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- 2 Body temperature  $26^{\circ}$
- 3 Body temperature  $22^{\circ}$
- 4 Body temperature  $39^{\circ}$

## PLATE 2

### EXPLANATION OF FIGURES

5 to 10 Sagittal sections taken in order passing from the right to the left sides of the brain stem of pigeon 126. Because of lack of detailed knowledge of the thalamic and mesencephalic nuclei and fiber tracts in the bird, these parts are not labeled. Camera-lucida drawings.  $\times 6$

*Cc*, cerebellum

*Hyp*, hypothalamus

*III*, oculomotor nerve

*M*, midbrain

*MO*, medulla oblongata

*N*, region of broken, unstained, apparently softened or necrotic tissue

*Opt ch*, optic chiasma

*OL*, optic lobe cortex

*ON*, optic nerve

*PC*, posterior commissure

*V*, ventricle of the optic lobe

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These words are very true, but still this danger did not make him refrain from expressing his thoughts on a subject that we all as biologists both love and fear—natural philosophy in the widest sense of the term

That this same danger threatens me, who not only consider memory, but also association and attention (concentration) as general functions of organized matter, is clear. It was therefore, not without some hesitation, that I sent this paper to the editor of *The Journal of Comparative Neurology*. Its title sounds a little bold in the ears of most biologists

I also thought it better at first to change 'logetic' in its title into 'logical'. Since, however, we are accustomed to consider logic reason, as something that is peculiar to conscious thinking, and there will be question here of a general principle of life, which, with other faculties but according to similar laws, also operates outside conscious thinking, I have preferred to use the word 'logetic' to indicate a broader idea of 'logos', formerly used to give expression to something that is more than that small part of reason of which we become conscious in our 'logical' thinking

I do not want to be misunderstood. I do not mean to say that logical 'thinking' accompanies the somatic development, nor that a tissue differentiation of the same form that obtains in the soma accompanies the building up of our spiritual life. No spiritualization of the somatic, therefore, nor a materialization of the spiritual. I only want to point out that one and the same principle of life, which Aristotle called 'psyche,'<sup>3</sup> with other faculties, but ruling with similar conformities, is peculiar to both, and leads in both to results which are different in effect, but which agree in

<sup>3</sup> So this is a psyche in a much wider sense than it has been used in the word 'psychology'. Cf. Hammond, *Aristotle's Psychology: A Treatise on the Principle of Life*. *De Anima*, book 1, chapter 5, Alinea 31. "parts of the soul are all found in every one of these bodily divisions and they are of like with each other and with the entire soul."

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and which are mostly indicated by the two centrosomes. So here, too, there are two (sometimes more) simultaneous centers of influence, which play an essential part in the accomplishment of the process.

Whereas, however, the simultaneous action of influences causes a 'linking,' an *association* in the construction of mental life and also of the nervous system, there appears a *differentiation* in the other case (in somatic development), a differentiation which, however, remains a unit, an individual, in other words, the 'linking' of the parts which is a consequence of the process in mental life, is present at the starting-point in somatic development and persists.

Speaking properly, however, it may be said, that here, too, the 'linking' of the results of those influences does not arise till the differentiation has been accomplished, because, when the germ-cell was still one cell, the influences, which bring about the differentiation, had not yet acted, and so (apart from engrammatic factors) the results of those simultaneous influences, too, could not as yet have been linked.

*So in both processes there are simultaneous influences, from which originates a formative process, in both a linking of those influences, in the cerebral linking, however, an integrated association of them and in the development of the germ-cell a differentiated association.* In both cases, however, there arises a construction, which is a product of correlated influences of the surroundings.

Let us consider in this light the influence of the medium on the differentiation of the cells. In order to explain how the division and multiplication of the cells is at the same time attended with a qualitative differentiation of the daughter-cells, it is supposed on good experimental grounds that the two sides of the mother cell, owing to their different situation—owing to the fact that they are exposed to different influences—do not undergo the same differentiation.<sup>6</sup> That from the same blastomeres entirely

<sup>6</sup> Wilson (also Driesch and Hertwig) "The relative position of the blastomere in the whole determines in general what develops from it, if its position be changed, it gives rise to something different, its prospective value is a function of its position." (The cell in development and inheritance.)

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In the ganglion-cells of the retina O v d Stricht found the centrosome in the dendritic part of the cells and so did N v d Stricht in young spinal ganglion cells still in the bipolar stage (In adult monopolar ganglion cells they seem to lie often near

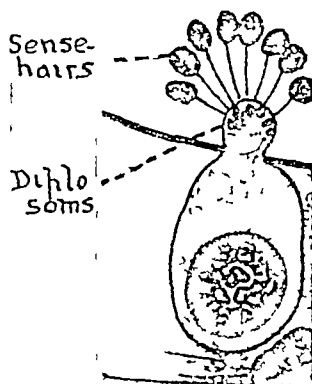


Fig 1 Sense cell of the saccus vasculosus of *Pleuronectes limanda*. After Dammerman

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This position of the centrosome near the place which receives the influences from the surroundings reminds us of the structure of the spermatozoid where it is attached to the flagellum, and the same applies to ciliated epithelium.

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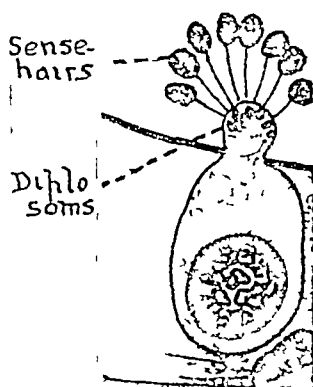


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a reciprocal action based on a striving after equilibrium, a contrary differentiation or manifestation of energy which can react toward a defect with a proliferation, toward pressure with increased hardness toward light with pigment, toward toxin with antitoxin<sup>8</sup>

Moreover the organism whose reciprocating energy has thus been evoked remains throughout a unit materially and functionally viz, it shows in all its parts an associated correlation, and the developed organism manifests itself as one correlated system whose harmony is astonishingly reasonable in a logetic sense

One need only think of the relation between lens, retina, and pigment in the eye, the mutual development of which far exceeds in logical, or rather logetic, relation the mental possibilities of our conscious logical intellect

Thus there is in our somatic development a logetically correlated relation which has its origin in the same cause as the mental associations, viz, in different but simultaneously operating i.e., correlated, influences

In this development of form the 'function' is inherent of which the 'logetical' relation with the surrounding world and with the rest of the body is not less evident, and operates with the exactness of mathematical reasoning, witness the different ways in which accommodation of vision is effected in the animal series

It appears, therefore, that the *associative differentiation of the body* is in its result a different thing from the *associative linking in the nervous system*, but that both of them find their origin in correlated stimuli

Both the neuromic linking and the building up of our conscious mental life, on the one hand, and bodily differentiation, on the other, are reasonable correlations, originating in correlated irritations, two different forms of logetic realization

Besides these, there are in both processes other common factors, which again manifest themselves in each in a different way,

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plete ego, which seems to be present in the nerve-cell as a derivative of the germ-cell, and from this it follows, not that 'the light is to be seen,' but that 'I' (i.e., the primitive many-in-oneness of potentialities) 'see this light' (cf. also Hughlings Jackson, Pick, and others)

All perceptions<sup>9</sup> and correlations always lie in this ego, which may represent the primitive many-in-oneness of mental life. In these perceptions, however, the ego stands in the background of one's consciousness. Indeed, it is not seldom in the first instance made active by a perception, which it precedes, however, in potentiality.

It seems now probable to me that here, too, this 'ego,' i.e., the direct experience of myself, the primitive unit, is bound to all nerve-cells, and that owing to this the consciousness of self (not the secondarily formed conception of myself) can remain, notwithstanding large destructions by illness, which it would not be possible to explain in an exclusively secondary linking of the different neurons in a very imperfect secondary 'ego.'

The secondarily integrated conscious image is very incomplete of its kind, and human ingenuity would require much more than a lifetime of observations and experiences to build up, in secondary integrations, all that which works as spiritually active factors in the individual ego.

The 'egoity' awaked by influences from without includes, however, undoubtedly, much more than lifetime experience and begins with a completion (be it un- or subconscious) which bears a perhaps infinite series of engrams and peculiarities, which in our subconsciousness are joined 'intuitively' (in an 'entelechië'<sup>10</sup> way).

<sup>9</sup> These perceptions preserve a certain separation because the interval also represents a situation of the ego.

<sup>10</sup> The word 'entelechiä,' first used by Aristotle, comes probably from 'enteles' (fulfilment, completion) and 'echein,' to have. It is in a way opposite to teleology. In teleologic functions the 'logos' of the 'telos,' the knowledge of the end (the aim) is present. In entelechië processes the character of the result develops through intrinsic forces and the result is only known when reached (unforeseen). An example of the latter is the development of man from ape-like ancestors, who could not have the man-like characteristics as an aim, since these did not yet occur at that time.

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### Actividad metabólica del sistema nervioso

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Las ratas estimuladas eléctricamente durante un periodo de 10 a 24 horas presentan en su cerebro una cantidad de nitrógeno no proteínico relativamente mayor que el de las ratas normales escogidas como término de comparación. Una estimulación semejante durante 6 horas no aumenta el contenido normal en la rata que "no lucha," pero las que luchan presentan un incremento de productos metabólicos en el cerebro. Las ratas que lucharon violentamente produjeron una cantidad considerable de nitrógeno no proteínico, aun después de una a cuatro horas de estimulación. Las que lucharon durante una hora presentan la cantidad normal de nitrógeno no proteínico en el cerebro después de 42 horas de descanso. El aumento de este nitrógeno en el cerebro como resultado de una lucha violenta, se interpreta como debido en parte a productos metabólicos, que resultan del aumento de la actividad fisiológica general del cuerpo, los cuales llegan al cerebro con la sangre, y, parcialmente también, como el resultado del aumento de la actividad metabólica del mismo cerebro.

Translation by Jose I. Noides  
Carnegie Institution of Washington

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eliminating as much as possible the factor of fear from the control rats. The rats were usually fed with a mixture of 'Uneda biscuit' and condensed milk, at about 9 A.M. In making the tests two male rats were put into a box which was constructed in the following manner. A wooden box about 11 inches long, 10 inches wide and 8 inches high was made, in the bottom of which numerous nails were placed with their tips just exposed on the inner surface of the bottom. These nails were connected by means of a copper wire and the ends of these wires were in turn connected with a battery, so that an electric current could pass through them. The rats standing in this box were stimulated for a period of three seconds in every two minutes by the passage of a current. The rats began to fight immediately or shortly after the electrical shock was given, as if one rat held the other responsible for the shock received. Sometimes the rats refuse to fight, and in such cases a light pricking of the tail with a sharp needle always provokes fight almost at once. When once started, the rats continued fighting under the stimulus of the electrical shock alone. Usually the two test rats were taken from different litters, because the rats which belong to the same litter and are accustomed to living in the same cage do not normally fight with each other.

When the rats were brought from the rat house, I put those of the same litter in two separate boxes, one control rat and one test rat in one box, another control and test rat in the other box. Rats of more than 120 days of age were chosen, because Hatu ('17) found that the amount of non-protein nitrogen shows very slight age alteration after the rats pass this age, while on the other hand the rats which are younger do not fight vigorously. Males only were used.

The rats may continue fighting vigorously for several hours. In this operation both rats stand on their hind feet and push each other with their front paws, holding their bodies erect and straight and their mouths almost touching each other. Every time a shock passes both squeak and each pushes the other strongly and they may even bite one another. In some cases the rats continue this performance for more than six hours, while

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conventional mark made by some other member of the laboratory and thus the non-protein nitrogen determinations were conducted in entire ignorance as to which flask belonged to the control or which to the test series, thus avoiding any personal bias in the determinations

### EXPERIMENT SERIES 1

These experiments have been made to see whether or not the amount of non-protein nitrogen in the brain is changed as the result of stimulation (fighting). Altogether six control and six test animals were used. The period of stimulation extended from ten to twenty-four hours. The rats did not fight at all in two cases and only slightly in one. In no instance was the method of pricking with a needle applied to induce fighting. During the experimental period both the control and test animals were not fed except with water. The results are shown in table 1.

As will be seen from table 1, the relative amount of non-protein nitrogen (per 100 grams) in the brain of the test rat is significantly greater than those given by the control rat. The amount of difference is greatest in the rats which had been stimulated for the longest period, but this may be mere coincidence, since the other two cases do not follow in this relation. The present results bring out at least two points. Since these rats were not fed during the period of stimulation, it is conceivable that the electrical shocks, although they did not induce actual fighting, might nevertheless through periodic irritation accelerate metabolic activity as compared with the rats which were not stimulated, and thus produce a form of mild inanition. It has been already found in my previous studies ('19) that during inanition (represented by the later part of the twenty-four-hour period) the non-protein nitrogen content of the brain shows some increase. It will be seen, however, from the later experiments that this increase in the non-protein nitrogen may be mainly due to stimulation, though inanition may also contribute to it.

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the nerve cells as the result of electrical stimulation, since we know from the work of previous investigators that the nerve cells show a definite alteration as the result of direct stimulation of peripheral nerves (Hodge, Dolley, and others)

We shall, however, reserve this discussion until further experimental data are presented

Whatever might be the real cause or causes, we see from this preliminary test that as the result of stimulation the amount of non-protein nitrogen in the brain increases. On account of some defects in our kymograph, it became impossible in the subsequent experiments to run the machine for long periods continuously, and in the later tests we were thus obliged to reduce the maximum stimulation period to six hours

## EXPERIMENT SERIES 2

In the present experiments the test rats were stimulated for six hours with a current from four batteries<sup>1</sup>. Some of these rats did not fight at all, while others made a good fight. When the data are arranged according to the amount of fighting, we obtain interesting results.

As will be seen from table 2, after six hours of stimulation those rats which fought give a significantly greater amount of non-protein nitrogen as compared with the controls, while those rats which did not fight give an amount of non-protein nitrogen almost identical with that for the control brains. It appears from these results that the electrical stimulation alone for a period of six hours is not sufficient to produce a greater accumulation of non-protein nitrogen in the brain, but an emotional disturbance does cause an excessive accumulation of the non-protein nitrogen.

The present experiment seems to indicate that an increased amount of non-protein nitrogen found in the brain of the rats which were stimulated for more than ten hours, and which did not fight at all (experiment 1) might be mainly due to a somewhat increased rate of metabolic activity of the test rats, thus producing a mild inanition. It seems from these data reasonable

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to conclude that as a result of great emotional disturbance the circulation of the blood is accelerated, and as a consequence the cell metabolism of the brain is also accelerated, thus producing a greater amount of metabolites in the brain

### EXPERIMENT SERIES 3

Thus far the test rats did not fight vigorously, owing to their lack of response to the electrical stimulus. We found later that when their tails are lightly pricked with a sharp needle they at once begin fighting. By such a simple procedure, accompanied by the electrical stimulus, the rats are made to fight severely, at the same time squealing and biting each other. When once such a violent fight starts the periodic shock is irritating enough to make the fight continue until one rat becomes exhausted and tries to avoid its opponent's attacks. The amount of non-protein nitrogen was determined for those rats which had such a very severe fight for from one to four hours. The results are given in table 3.

The results obtained from the eight independent experiments, using sixteen test rats, show clearly that the amount of non-protein nitrogen in the brain increases as the result of severe fighting when compared with that obtained from the control brain. The amount of non-protein found is, however, irregular and there is no precise indication of a proportional increase with prolongation of the fighting period. In fact, in one instance (the third in table 3) a large amount of decrease is shown as the result of severe fighting for three hours. This decrease in the amount of non-protein nitrogen might have been the result of a complete exhaustion. These irregularities in the amount of non-protein nitrogen found in the brains of test animals may be due to the fact that there are considerable individual differences as to the behavior during experimentation. Some rats are very aggressive and may continue violent fighting without cessation, while there are instances in which the rats fight severely for a few seconds, then stop fighting for some time, only to resume again. Still more important in accounting for the irregularities is the

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fact that some rats show physical exhaustion much quicker than others. It seems to be clear also from the present data that this increase in the amount of non-protein nitrogen in the test brain cannot be the result of inanition, since the period of stimulation is only from one to four hours—mostly one hour—and indeed the increase is often more marked with rats which were stimulated for one hour only.

We may conclude, then, that as the result of violent fighting the amount of non-protein nitrogen accumulates far in excess of that in the control brain although the exact cause for such an increase is still to be carefully considered.

#### EXPERIMENT SERIES 4

The experiments so far show clearly that the amount of non-protein nitrogen in the brain increases as the result of stimulation, and it was now thought desirable to determine the effect of rest on the content of the metabolites. For this purpose the rats were induced to fight violently for one hour by methods already described. After the lapse of this period, the test rats were placed separately in the usual laboratory cages and kept there with abundant food and water for from twenty-four to forty-two hours. The results of recuperation for these periods are shown in table 4.

From table 4 it is clear that the amount of non-protein nitrogen in the brain of rats which have rested for twenty-four hours is still significantly higher than those in the control brain. However, in the rats which have rested for forty-two hours the relative amount of non-protein nitrogen is almost the same in both the control and test animals, though the test brains still give a slightly higher value. We might conclude from these data, therefore, that for full recovery to the normal state the rats which have fought violently for one hour require more than forty-two hours' rest. I regret that I cannot extend the observations on resting rats, owing to the limitations of my stay in this country.

fact that some rats show physical exhaustion much quicker than others. It seems to be clear also from the present data that this increase in the amount of non-protein nitrogen in the test brain cannot be the result of inanition, since the period of stimulation is only from one to four hours—mostly one hour—and indeed the increase is often more marked with rats which were stimulated for one hour only.

We may conclude, then, that as the result of violent fighting the amount of non-protein nitrogen accumulates far in excess of that in the control brain although the exact cause for such an increase is still to be carefully considered.

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## DISCUSSION

From the data presented it seems clear that as the result of severe fighting the amount of non-protein nitrogen increases considerably in the brain. The interpretation of this phenomenon is difficult. In association with violent fighting there is more or less physical exercise, which necessarily accompanies fighting, and we should anticipate an effect of fatigue and whatever changes such fatigue may produce on the brain. Because great emotional disturbance is necessarily associated in this case with marked bodily activity, the greater amount of non-protein nitrogen found in the brain in the present experiment might be considered a result of abnormal physiological activity of various organs and tissues, besides that of the nervous system itself.

The sources of non-protein nitrogen in the central nervous system are two, one is that of the metabolites transported to the brain by means of the blood, and the second is the production of metabolites by the nervous tissue itself. It is, however, impossible to determine from the present experiments alone which of these sources should be held more largely responsible for the greater accumulation of the metabolites in the brain. It is, however, true that the greater activity of the muscles and organs during severe fighting must increase the amount of metabolites in general, and at the same time we are also justified in concluding that the brain tissue itself must increase in its activity. This latter conclusion follows from the investigations of Hodge ('92), which showed that conspicuous structural alterations of the spinal ganglion cells follow the direct electrical stimulation of peripheral nerves. Hodge further demonstrated that the cells of spinal ganglia of English sparrows, of the cerebrum of pigeons, and cerebellum of swallows and antennal lobes of bees obtained at the end of the day, that is, after a period of activity, show structural changes as compared with those obtained at the beginning of the day, or after a night of rest.

Similar observations were made subsequently by several observers, and we may mention here the work of Mann ('95) on the motor cells of the spinal cord and cells of the retina as one illustration.

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Resumen por los autores, Mathilde L Koch y Oscar Riddle

## Nuevos estudios sobre la composición química de los cerebros de palomas normales y atáxicas

Una segunda series de análisis de cerebros de palomas afectadas de una falta hereditaria de regulación en los movimientos voluntarios, demuestra que estos cerebros se distinguen del cerebro normal por el tamaño y composición química. Los cerebros de las palomas atáxicas son mas pequeños. Los autores han hecho ocho análisis de la parte anterior (cerebro) y posterior (cerebro-médula) del encéfalo. Cuatro de estos análisis se llevaron a cabo en palomas atáxicas y los otros cuatro en aves normales de una edad comparable. Los cambios químicos encontrados están más pronunciados en los cerebros de las palomas fuertemente atáxicas que en los de las menos afectadas. También han hecho análisis adicionales de los encéfalos completos de aves muy jóvenes y muy viejas. Los datos sobre los cambios químicos del cerebro que acompañan a la edad han sido obtenidos para una serie de individuos de diversas edades en la paloma. Estos cambios son paralelos a los observados previamente en el hombre y la rata. El examen de esta "serie de edad" más extensa de cerebros de palomas les ha permitido evaluar mucho mejor que en su trabajo precedente la relación entre las diversas fracciones químicas y la edad. Las diversas fracciones de fósforo y azufre lipoide parecen variar en consistencia con la edad hasta los 600 días. Una revisión de la significación de los resultados obtenidos en la presente serie de análisis y en la precedente, conduce a la conclusión de que las diferencias observadas indican una escasa diferenciación química o relativa falta de madurez de los cerebros atáxicos. La diferenciación química, que probablemente incluye en parte la mielinización, no procede aparentemente tan deprisa en el encéfalo y, más particularmente, en el cerebelo-médula de los individuos atáxicos como en el encéfalo de los individuos normales.

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### MATERIALS AND METHODS

The brains used in the preparation of samples I, II, III, IV, and VI are from birds similar to those used in our previous study except for age differences The two groups of ataxic birds showed the disorder to different degrees The older group (sample III) being clearly the more affected<sup>3</sup> The birds which supplied the material for sample VI were considerably younger than the birds used in the earlier study, while the other four samples were obtained from somewhat older birds (II and IV), and from much older (I and III) birds The birds used in the preparation of sample VI were mostly too young to classify as normal or ataxic

All of the above-mentioned birds, like those used for the previous study, were birds descended from the first obtained ataxic or affected individual These birds, ataxics and normals, were therefore considerably inbred The normals or 'controls' of these groups were of the same strain and parentage as the ataxics, they were, in the main, brothers and sisters of the ataxics Sample V contained the brains of the oldest common pigeons (mostly homers) of the same general kind, but without ataxic blood, which we could obtain from our collection

In the present study the cerebrum was analyzed separately from the cerebellum-medulla in four cases, i e, four groups of

<sup>2</sup> Precise information of this sort has been obtained hitherto, so far as we are aware, only in man and in the rat The data for man are very incomplete

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the present study are given in tables 1 to 3. The weights of the two parts of the brain, the weight of the entire brain, the weight of body, the sex, and the age of the birds of the group are included in the same tables. In these tables the birds are

TABLE 1

*Details on the materials used in the preparation of the normal (control) pigeons' brains*

	NUMBER OF BIRD	SEX	BODY WEIGHT	BRAIN WEIGHT			AGE
				Cere- bellum and medulla	Cerebrum	Whole brain	
Older normals (sam- ples I and Ia)	B530	♂	307	0 445	1 435	1 880	887
	B523	♂	352	0 457	1 563	2 020	820
	B548	♂	344	0 465	1 500	1 965	783
	B665	♂	376	0 466	1 413	1 879	722
	B489	♂	372	0 460	1 423	1 883	674
	K288	♂	315	0 450	1 518	1 968	564
	K112	♀	315	0 395	1 385	1 780	432
	K178	♂	340	0 505	1 471	1 976	414
	K217	♀	305	0 462	1 364	1 826	392
	E232	♂	304	0 447	1 406	1 853	298
Average			334 0	0 4552	1 4478	1 9030	598 6
Younger normals (samples II and IIa)	K251	♀	225	0 418	1 360	1 778	294
	K239	♀	316	0 403	1 418	1 821	290
	K284	♂	291	0 482	1 573	2 055	281
	K235	♀	288	0 465	1 374	1 839	262
	K265	♀	293	0 435	1 333	1 768	255
	K250	♂	313	0 492	1 486	1 978	219
	M364	♂	291	0 503	1 400	1 903	169
	M366	♂	352	0 520	1 507	2 027	129
	M471	♂	322	0 487	1 443	1 930	80
	M430	♀	274	0 420	1 358	1 778	76
Average			296 5	0 4625	1 4252	1 8877	205 5

all arranged according to decreasing age. These tables are given in order that all of the necessary data may be presented, including the size and composition of each sample as prepared for analysis. These tables are not used directly in the comparisons made below.

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Indeed, in the four really comparable<sup>5</sup> groups the lowest average brain weight for males is higher than the highest average for females. The same is true for the two separate parts of the brain—the male cerebellum-medulla is larger and the male cerebrum is larger.

That this larger size of the brain, and of both parts of the brain, of the male is not wholly dependent upon the larger body size of the male is indicated by the fact that two only of the male

TABLE 3  
*Details on the materials used in the preparation of groups V and VI*

	NUMBER OF BIRD	SEX	BODY WEIGHT	BRAIN WEIGHT			AGE
				Cerebellum and medulla	Cerebrum	Whole brain	
Older pigeons of other strains (sample V)	H-A	♂	435	0 438	1 527	1 965	3266
	H-B	♂	394	0 511	1 535	2 046	3264
	169	♀	334	0 510	1 528	2 038	1287
	A21	♀	335	0 472	1 523	1 995	1238
	A313	♀	258	0 457	1 505	1 962	1048
Average			351	0 4776	1 5236	2 0012	2021
Younger pigeons of the ataxic strain (sample VI)	M475	♀	316	0 442	1 352	1 794	59
	M452	♀	163	0 393	1 223	1 616	59
	M478	♂	255	0 454	1 384	1 838	52
	M479 <sup>1</sup>	♂	319	0 427	1 323	1 750	50
	M441	♀	264	0 367	0 941	1 308	37
	M408	♀	129	0 335	0 940	1 275	35
	M415	♀	75	0 198	0 542	0 740	22
Average			217 3	0 3737	1 1007	1 4744	45

<sup>1</sup> Known to be ataxic

groups are larger and two are smaller than the associated females. For the mature birds the brain weight shows considerable independence of body weight.

The relation of age to the size of the brain and to each of the two parts into which we have divided it can be partly under-

[<sup>5</sup> Group VI is clearly immature, group V is not of the same strain. In the latter group, moreover, the disparity of age is extreme, the males being very old (nine years) and the females in their prime (three years)]

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stood from a study of the data of table 4 and partly from the data previously obtained by us ('18, table 3, in part reproduced here, table 8). It is clear that neither females of 42 days nor males of 51 days (averages on table 4) have fully developed brains. Two females of 69 and 127 days ('18, table 3), however, each had a brain nearly as large (1 811 grams and 1 813 grams) as that of the largest female brain of groups I and II (K235 = 1 839 grams, age 262 days, table 1) and larger than the average brain (1 803 grams and 1 797 grams, table 4) of the females of these much older normal groups. Similarly, a male of 124 days ('18, table 3) had a brain larger (1 943 grams) than the average (1 928 grams) of eight normal males of 645 days (average, table 4).

It is reasonably clear that in this particular strain of birds the maximum brain weight is usually attained not much later than 100 days after the beginning of development (eighteen days for incubation). In all of our present and previous analyses of pigeon brains (table 8), therefore, only the brains of group VI of the present series were undersized because of age. Groups II and IV, which are compared with each other, have each two birds aged less than 100 days.

#### *Relation of ataxia to brain size*

The relation of brain size to normality and ataxia may now be confidently studied, since the influence of sex, body weight, and age have already been considered. Four quite comparable groups (I to IV, table 4) are available, two of these are brains from normal birds and two from ataxics, and there are both males and females in each of the four groups for comparison. The following is found:

The *whole brain* of each of the ataxic groups is smaller<sup>6</sup> than that of either of the two normal groups (tables 1 and 2). The

<sup>6</sup> There is a high percentage of males in one normal group and a high percentage of females in one ataxic group which considerably affects the brain size of these two. But the comparison between the normal and ataxic males of these two groups, and between the normal and ataxic females of these two groups, is just as valid as are the similar comparisons between the other two groups in which no disparity of sex exists. The mean weights of the various groups permit a quite fair comparison from one group to another.

stood from a study of the data of table 4 and partly from the data previously obtained by us ('18, table 3, in part reproduced here, table 8). It is clear that neither females of 42 days nor males of 51 days (averages on table 4) have fully developed brains. Two females of 69 and 127 days ('18, table 3), however, each had a brain nearly as large (1 811 grams and 1 813 grams) as that of the largest female brain of groups I and II (K235 = 1 839 grams, age 262 days, table 1) and larger than the average brain (1 803 grams and 1 797 grams, table 4) of the females of these much older normal groups. Similarly, a male of 124 days ('18, table 3) had a brain larger (1 943 grams) than the average (1 928 grams) of eight normal males of 645 days (average, table 4).

It is reasonably clear that in this particular strain of birds the maximum brain weight is usually attained not much later than 100 days after the beginning of development (eighteen days for incubation). In all of our present and previous analyses of pigeon brains (table 8), therefore, only the brains of group VI of the present series were undersized because of age. Groups II and IV, which are compared with each other, have each two birds aged less than 100 days.

#### *Relation of ataxia to brain size*

The relation of brain size to normality and ataxia may now be confidently studied, since the influence of sex, body weight, and age have already been considered. Four quite comparable groups (I to IV, table 4) are available, two of these are brains from normal birds and two from ataxics, and there are both males and females in each of the four groups for comparison. The following is found:

The *whole brain* of each of the ataxic groups is smaller<sup>6</sup> than that of either of the two normal groups (tables 1 and 2). The

<sup>6</sup> There is a high percentage of males in one normal group and a high percentage of females in one ataxic group which considerably affects the brain size of these two. But the comparison between the normal and ataxic males of these two groups, and between the normal and ataxic females of these two groups, is just as valid as are the similar comparisons between the other two groups in which no disparity of sex exists. The mean weights of the various groups permit a quite fair comparison from one group to another.

direction of the normal female It will be pointed out later that ataxia is found more often in females than in males Since the observed effects of ataxia on the male all take the direction of the female, it may be asked, does this fact have any bearing upon the predominant appearance of the derangement in female offspring?

Before concluding the above considerations (in which the materials entering into the composition of the samples are being considered as fully as a paper presenting chemical data permits), emphasis may be placed upon the fact that samples I-Ia and III-IIIa (older normals and older ataxics), though quite comparable as to age, are not so in regard to sex Also, that this sex difference at least partially accounts for the size differences of the brains of these two groups And, further, that differences of brain size may be of significance in the results of the chemical analysis Donaldson ('16) obtained from the rat evidence "that both the relative and absolute weight of the brain \* \* \*, at a given age, are factors tending to modify the percentage of water present, in the sense that the heavier brain or cord shows the smaller percentage of water" Donaldson<sup>7</sup> also indicates that in a given species the larger (heavier) brains at a given age tend to have a higher percentage of white substance On this basis the larger brains of both the older and younger normals (samples I-Ia, II-IIa) might be expected to show lower water values (and other chemical evidences of greater age) than the ataxic groups of somewhat smaller brain size with which they are compared Possibly such size relations do slightly influence the amount of the various chemical fractions obtained by us We would note, however, that the cerebrum of the younger normals (IIa) and the younger ataxics (IVa) were of equivalent (total) size (14 252 grams and 14 233 grams, tables 1 and 2), and in each sample the sexes were equally represented, nevertheless, when the figures obtained for these two groups are compared, on the basis of the nine constituents found to be characteristic of age in this series (p 98), it is found that six of these nine constituents here indicate the relative immaturity of the ataxic

<sup>7</sup> Personal communication

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phosphatids) which are really indicative of age in very young brains have very limited or quite uncertain values when applied to brains of some of the older ages. This particularly applies to several ages actually studied by us. It is, therefore, necessary to restate here the chemical criteria for differentiating younger and older stages, as this applies to the pigeon brain for those particular ages which we are now to compare.

The data given in table 8 require the following conclusions.

*Water* is decreased relative to solids throughout the entire age series. It is true that the moisture figures obtained for the several groups of normals do not correctly indicate the age of the group in all cases. For example, the normal brain of 106 days is shown to have a slightly lower percentage of water than the normal brains of 205 days, and an ataxic of 133 days slightly less water than an ataxic of 206 days. Ataxia itself probably further complicates the smoothness of the figures for the series as a whole. Nevertheless, a general tendency to a decrease of water with increasing age is unquestionable.<sup>8</sup>

*Protein* plainly decreases with increased age. Only the figure for the normals of 106 days breaks the complete smoothness of the curve for the entire series of normals.

*Lipoids* increase with increased age, although the figures actually obtained are not wholly consistent, neither for the normals considered alone nor for the ataxic series. In fact, between 106 days and 598 days very little change is indicated in the amount of lipoids. This doubtless indicates that myelination is practically completed in these pigeons at 106 days.

*Extractives* are present in the solids in greater amount in the forty-five day brain than at any other time. In normals of 106 days, however, no more extractives are present than in normals of 2,021 days, and less is found than in normals of 205 and 598

<sup>8</sup> It should be borne in mind that there is opportunity for error in the moisture estimation of any organ such as the brain. First, through unequal evaporation from the brain surface during the preparation of the sample, and, second, through the presence of unequal quantities of blood within the organ when weighed. Whether ataxia itself offers any special complication is at present unknown to us. Again, any loss or gain to the solids of either the alcohol-ether soluble or alcohol-ether insoluble fraction would serve to modify the recorded amount of moisture.

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above-stated abnormal relation of phosphatids and sulphatids to age (at 200 to 600 days) disappears. The difference of result on this basis of calculation is directly due to the fact that total

TABLE 5

*Chemical composition of cerebrum and cerebellum-medulla of normal and ataxic pigeons (in per cent of solids)*

GROUP	CEREBRUM				CEREBELLUM AND MEDULLA			
	IIa Normals 205 days	IVa Ataxics 200 days	Ia Normals 598 days	IIia Ataxics 600 days	II Normals, 205 days	IV Ataxics 200 days	I Normals 598 days	III Ataxics 600 days
	Younger		Older		Younger		Older	
Water in per cent	81.0	80.3	79.7	79.6	77.9	78.0	77.9	77.8
Proteins	52.1	51.3	49.9	50.3	46.3	46.8	45.8	46.2
Lipoids	34.4	35.3	35.8	35.0	40.7	41.4	41.9	41.8
Extractives	13.5	13.4	14.3	14.7	13.0	11.8	12.3	12.0
Cholesterol	7.5	7.4	7.4	7.3	8.8	8.8	9.0	8.9
Phosphatids	22.5	22.5	22.3	20.2	24.6	23.4	22.5	23.0
Sulphatids	8.4	8.4	6.8	5.6	13.3	11.3	11.8	15.9

*Distribution of sulphur in per cent of total sulphur*

Protein-sulphur	65.3	69.7	66.6	69.9	55.8	54.5	56.8	49.6
Lipoid-sulphur	18.1	16.5	18.9	15.3	26.6	23.5	27.2	32.1
Extractive-sulphur	16.6	13.8	14.5	14.8	17.6	22.0	16.0	18.3
Total sulphur (in per cent of solids)	0.93	1.02	0.72	0.76	1.00	0.96	0.87	0.97

*Distribution of phosphorus in per cent of total phosphorus*

Protein-phosphorus	17.7	20.1	13.9	16.0	18.7	18.0	17.9	20.4
Lipoid-phosphorus	63.0	59.8	67.5	62.1	61.7	60.2	61.2	59.3
Extractive-phosphorus	19.3	20.1	18.6	21.9	19.6	21.8	20.9	20.3
Total phosphorus (in per cent of solids)	1.39	1.46	1.28	1.26	1.55	1.50	1.43	1.51

phosphorus and total sulphur are reduced in the brains of about 600 days. Calculated thus, lipoid-phosphorus is in greater amount in the 598-day normal than in the 205-day normal.

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TABLE 5

*Chemical composition of cerebrum and cerebellum-medulla of normal and ataxic pigeons (in per cent of solids)*

GROUP	CEREBRUM				CEREBELLUM AND MEDULLA			
	IIa	IVa	Ia	IIa	II	IV	I	III
	Normals	Ataxics	Normals	Ataxics	Normals,	Ataxics	Normals	Ataxics
	205 days	200 days	598 days	600 days	205 days	200 days	598 days	600 days
	Younger		Older		Younger		Older	
Water in per cent	81.0	80.3	79.7	79.6	77.9	78.0	77.9	77.8
Proteins	52.1	51.3	49.9	50.3	46.3	46.8	45.8	46.2
Lipoids	34.4	35.3	35.8	35.0	40.7	41.4	41.9	41.8
Extractives	13.5	13.4	14.3	14.7	13.0	11.8	12.3	12.0
Cholesterol	7.5	7.4	7.4	7.3	8.8	8.8	9.0	8.9
Phosphatids	22.5	22.5	22.3	20.2	24.6	23.4	22.5	23.0
Sulphatids	8.4	8.4	6.8	5.6	13.3	11.3	11.8	15.9

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Protein-phosphorus	17.7	20.1	13.9	16.0	18.7	18.0	17.9	20.4
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*Protein-phosphorus* decreases wholly consistently with age in all of the normals. *Extractive-phosphorus* decreases progressively with age. This rule fails, however, in the very old (2,021-day) brain. *Total phosphorus* also progressively decreases with age.

In the comparison of the normal and ataxic brains, the younger age is characterized, therefore, by higher values for water, protein, protein-phosphorus, extractive-phosphorus, and total phosphorus, and by lower values for lipoids, cholesterol, lipid-phosphorus, and lipid-sulphur. A comparison, on the basis of these nine constituents, of corresponding parts of the brain of normals and ataxics will be made first. That of the whole brain of all of the normals and ataxics can be better done later.

*Results of analysis of cerebrum and cerebellum-medulla of normals and ataxics (table 5)*

The cerebrum of the younger (less) ataxic group gave lower figures for moisture, protein (extractives),<sup>12</sup> cholesterol, lipid-sulphur, and lipid-phosphorus than the younger normals with which they should be compared. Higher figures were obtained for lipoids, protein-phosphorus, extractive-phosphorus, and total phosphorus. Six of these figures indicate that the cerebrum of the younger ataxics (206 days) were less differentiated than those of the younger normals (205 days), three figures point to the opposite conclusion.

The cerebellum-medulla of the younger ataxics show smaller values for lipid-sulphur, protein-phosphorus, lipid-phosphorus (phosphatids, sulphatids, extractives), and total phosphorus, greater values for moisture, protein, lipoids, and extractive-phosphorus. Five of these figures point to the (less) ataxic cerebellum-medulla as the younger stage, while three are opposed. Cholesterol shows no difference. Summarizing this comparison of parts of the brain of younger normals and younger (less) ataxics, it may be said that the results show but little of chemical

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ferentiated, or less old, than are these parts of the brain in normals of equivalent age. Further, the evidence obtained from the older strongly ataxic brains is more decisive than that obtained from the younger less ataxic brains.

*Distribution of sulphur and phosphorus in cerebrum and cerebellum-medulla*

In table 6 are given the data on the distribution in cerebrum and cerebellum-medulla of sulphur and phosphorus calculated in per cent of solids. That method of calculation scarcely changes<sup>13</sup> the description already given above in terms of total sulphur and total phosphorus. Particular attention may be directed only to differences in distribution of these elements in the cerebrum and cerebellum-medulla. These data are the first thus far obtained for any bird.

Protein-sulphur is more abundant in the cerebrum than in the cerebellum-medulla. Lipoid-sulphur and extractive-sulphur is distinctly less in the cerebrum. The older birds (598 and 600 days) have markedly less sulphur in all fractions of the cerebrum than have the younger birds (205 and 206 days). In the cerebellum-medulla there is less of difference due to age. This probably indicates that the maximum sulphur content of the pigeon cerebrum is reached at nearly 206 days and thereafter decreases in relative amount (table 7). The sulphur of the cerebellum-medulla suffers no marked decrease during this period (206 to 600 days). Most of the sulphur of cerebrum and cerebellum-medulla is protein-sulphur.

The phosphorus of both the cerebrum and the cerebellum-medulla is chiefly lipoid-phosphorus. Protein-phosphorus and extractive-phosphorus are present in almost equal quantity in both parts of the brain. All three fractions of phosphorus are

<sup>13</sup> Only two of the figures compared above show a different relation to each other under the two methods of calculation. These occur in the lipoid-phosphorus and extractive-phosphorus of the cerebellum-medulla of the older ataxic group. Both became higher in the ataxic than in the normal. The numerical result is the same as before. Six figures still indicate the relative immaturity of the organ and three figures are opposed.

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readily compared (figures for both placed in *italics*) with the brains of similar ages. It is in these two series in which the abnormality was most marked that the clearest evidence for a chemical under-differentiation or relative immaturity of the ataxic brains is found. It is notable that in both of these groups the amount of water is either equivalent to or more than is indicated for their actual age, protein is present in excess in both, lipoids are deficient in both, cholesterol is lowest in both, phosphatids and sulphatids<sup>14</sup> are also low in both, total phosphorus

TABLE 8

*Chemical composition of the whole brain of normal and ataxic pigeons (in per cent of solids) Arranged according to age*

NUMBER	DESCRIPTION OF GROUPS	AVERAGE			WATER	SOLIDS			CHOLESTEROL	PHOSPHATIDS	SULPHATIDS
		Age	Body weight	Brain weight		Proteins	Lipoids	Extractives			
		<i>days</i>	<i>grams</i>	<i>grams</i>							
1	Normal <sup>1</sup>	2021	351	2.001	78.4	47.4	39.4	13.2	8.1	22.3	9.9
2	Ataxic	600	309	1.799	79.3	49.2	36.8	14.0	7.7	20.8	8.0
3	Normal	598	334	1.903	79.3	48.8	37.4	13.8	7.8	22.4	8.0
4	Ataxic	206	354	1.852	79.8	50.2	36.8	13.0	7.8	22.7	9.1
5	Normal	205	297	1.888	80.2	50.6	36.0	13.4	7.8	23.1	9.8
6	Normal	183	362	1.879	79.8	50.7	37.1	12.2	7.5	23.5	6.3
7	Ataxic	166	314	1.784	79.5	49.7	37.2	13.1	7.4	23.0	6.1
8	Ataxic	158	326	1.789	80.2	52.1	34.9	12.9	6.8	21.9	6.3
9	Ataxic	133	331	1.900	79.6	50.9	36.4	12.7	7.1	22.4	(8.1)
10	Normal	106	360	1.922	80.0	50.0	36.8	13.2	7.0	22.9	6.5
11	Mixed	45	217	1.475	82.6	51.9	33.7	14.4	6.5	22.8	4.1

<sup>1</sup> Birds not of ataxic strain, but of nearly similar variety

<sup>\*</sup> A mixed group, probably normals and ataxics, all from ataxic strain

NOTE—Nos 1 to 5 and 11 are new data, nos 6 to 10 are our earlier data ('18)

is low in both, extractive-phosphorus and protein-phosphorus are high in at least one case. In all of these fractions these two ataxic brain groups are less differentiated chemically than brains of their calendar age should be. Extractives are not distinctive of age for the ages actually considered and one ataxic shows a high the other a low figure for this fraction.

It thus appears that of those nine chemical fractions (eighteen for the two groups) which can be relied upon to reflect age differ-

<sup>14</sup> Confirmed by lipid-phosphorus and lipid-sulphur, table 7

readily compared (figures for both placed in *italics*) with the brains of similar ages. It is in these two series in which the abnormality was most marked that the clearest evidence for a chemical under-differentiation or relative immaturity of the ataxic brains is found. It is notable that in both of these groups the amount of water is either equivalent to or more than is indicated for their actual age, protein is present in excess in both, lipoids are deficient in both, cholesterol is lowest in both, phosphatids and sulphatids<sup>14</sup> are also low in both, total phosphorus

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4	Ataxic	206	354	1 852	79.8	50.2	36.8	13.0	7.8	22.7	9.1
5	Normal	205	297	1 888	80.2	50.6	36.0	13.4	7.8	23.1	9.8
6	Normal	183	362	1 879	79.8	50.7	37.1	12.2	7.5	23.5	6.3
7	Ataxic	166	314	1 784	79.5	49.7	37.2	13.1	7.4	23.0	6.1
8	Ataxic	158	326	1 789	80.2	52.1	34.9	12.9	6.8	21.9	6.3
9	Ataxic	133	331	1 900	79.6	50.9	36.4	12.7	7.1	22.4	(8.1)
10	Normal	106	360	1 922	80.0	50.0	36.8	13.2	7.0	22.9	6.5
11	Mixed	45	217	1 475	82.6	51.9	33.7	14.4	6.5	22.8	4.1

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<sup>\*</sup> A mixed group, probably normals and ataxics, all from ataxic strain

NOTE—Nos. 1 to 5 and 11 are new data, nos. 6 to 10 are our earlier data ('18)

is low in both, extractive-phosphorus and protein-phosphorus are high in at least one case. In all of these fractions these two ataxic brain groups are less differentiated chemically than brains of their calendar age should be. Extractives are not distinctive of age for the ages actually considered and one ataxic shows a high the other a low figure for this fraction.

It thus appears that of those nine chemical fractions (eighteen for the two groups) which can be relied upon to reflect age differ-

<sup>14</sup> Confirmed by lipid-phosphorus and lipid-sulphur, table 7

Our data concerning the localization of the derangement in the brain are still imperfect, because in our analyses the brain was separated into anterior and posterior parts only. It has been made clear that the chief size reductions occur in the posterior brain, and the evidence indicates that the deviations in chemical composition are accentuated in this same region.

Whether analyses of medulla and cerebellum separated from each other would have shown that all of the size and chemical changes occurred in one only of these organs is a question quite unanswered by our data. Nevertheless, the fact that changes were also found in the cerebrum would seem to indicate that the derangement is not absolutely confined to either of the chief divisions of the brain. It is possible, however, that localized affected areas are present and that these were 'diluted' by much normal material in our samples as prepared for analysis. If this were true, these particular localized areas would necessarily have a much greater degree of chemical under-differentiation than is indicated by the figures obtained by us.

The sex of the ataxic birds deserves a further statement. Those who may have carefully examined the character of the samples obtained from ataxic pigeons, in both the earlier and present series, will have noted that more female brains than male brains are found in these samples as prepared for analysis. In the earlier series (of ataxics) the proportion was ten females to five males, in the present series twelve females to eight males. This disproportionate representation of the two sexes in these samples was not consciously effected by us, since the sex of most of the individuals selected for the purpose was not known until after the birds were killed. In most cases they were selected chiefly because they were ataxic in one or another degree. Equality of the sexes was desired in our present samples, but could not always be obtained.

The excess of females in the two series of ataxics has led us to examine a segment of the breeding data in an effort to learn whether the ataxia more often occurs in females than in males. The data given below were obtained from a tabulation<sup>15</sup> of the

<sup>15</sup> All groups of offspring of ataxic blood or strain were included in the summary. The matings which had yielded no ataxic offspring were excluded.

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this table These show that (from the standpoint of relative amounts of the various chemical constituents) the cerebellum-medulla of the pigeon is chemically an intermediate of the pigeon cerebrum and the human brain (both of cerebrum and of cerebellum-medulla) Only the sulphatids of the seven fractions from the pigeon cerebellum-medulla fail to take an intermediate place between the pigeon cerebrum and human cerebrum

TABLE 9

*Comparison of the chemical composition of the adult cerebrum and cerebellum-medulla of man and of the pigeon (in per cent of solids)*

	WATER IN PER CENT	PRO- TEINS	EX- TRACT- TIVES	LIPIDS	PHOS- PHA- TIDS	CHOLE- STEROL	SUL- PHA- TIDS	CERE- BROS- IDES
(Part 1)								
Cerebrum								
Human <sup>1</sup>	76.9	37.7	7.9	54.4	28.3	10.0	9.6	6.6
Pigeon <sup>2</sup>	80.3	51.0	13.9	35.1	22.4	7.5	7.6	— <sup>3</sup>
Cerebellum-medulla								
Human <sup>1</sup>	78.1	40.4	8.7	50.9	25.0	6.4	9.0	7.4
Pigeon <sup>2</sup>	77.9	46.1	12.6	41.3	23.5	8.9	12.5	— <sup>3</sup>
(Part 2)								
Rearrangement of above figures in terms of decreasing (ontogenetic) age								
Human cerebrum	76.9	37.7	7.9	54.4	28.3	10.0	9.6	6.6
Human cerebellum	78.1	40.4	8.7	50.9	25.0	6.4	9.0	7.4
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<sup>1</sup> Average of two analyses by Koch and Voegtlin ('16) of cerebrum and cerebellum-medulla

<sup>2</sup> Average of two analyses (I-Ia and II-IIa of this paper)

<sup>3</sup> Cerebrosides have not been determined in the pigeon brain

If, now, the figures found in part 1 of table 9 be arranged in such an 'age series' as was prepared for the several pigeon brains of various ages (table 8), the result may be seen in part 2 of table 9. According to the places taken by cerebrum and cerebellum-medulla of man and the pigeon in this arrangement, the human cerebrum would seem to be the oldest—i.e., the most fully differentiated 'brain tissue,' the human cerebellum-medulla next in order, the pigeon cerebellum-medulla next. The pigeon cere-

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*The 'age series' of pigeon brains*

It has earlier been stated that it is only on the brains of man and the rat that we have had fairly adequate data for the progressive change of the various proximate chemical constituents during growth, or, more properly, as related to growth and age. The present work supplies such an 'age series' for the pigeon brain, and this series is now as extensive as are those now known for man and the rat. Each of these latter series includes observations on one or more relatively younger stages than we have studied in the pigeon. On the other hand, the data for the pigeon include one relatively older stage than has been obtained on either of the other two forms.

Except for differences which appear because of a lack of parallelism of age, the three 'age series' show that quite the same course of chemical differentiation is followed in the brain of man, the rat, and the pigeon. It is not our purpose to discuss these three series here. The essential similarity of results obtained on material from sources so unlike should, however, be noted as additional evidence for the trustworthiness of the methods developed by W. Koch ('09) for brain analysis. The brains of the three 'age series' mentioned above have all been analyzed according to Koch's method.

Since the above was written, we have had an opportunity to learn something of the results of the neurological studies made by Hoshino ('19) of the brains of some of this same family of ataxic pigeons. Although the present study was completed and fully described before we were aware of Hoshino's results,<sup>17</sup> it seems well to add here that the neurological and chemical studies support an essentially similar view. The bearing of Hoshino's summary statement is self-explanatory: "This may be regarded as a hypoplasia or developmental inhibition in the proprioceptive system, part of the motor system, and some structures connecting the medulla oblongata and cerebellum, occurring during growth, with scarcely any definite degeneration or secondary increase of neuroglia tissue."

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7 The significance of the results obtained in the present and former series of analyses has been reviewed. The evidence warrants the conclusion that chemical differentiation, probably represented largely by the relative abundance of the myelin, does not proceed as rapidly in the brain, and more particularly in the cerebellum-medulla, of ataxic birds as in the brain of normal birds.

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El autor ha estudiado funcional y anatómicamente cuatro palomas que presentaban ataxia hereditaria y tres palomas normales de la misma familia, las cuales le fueron enviadas por la Estación de Evolución Experimental de la Institución Carnegie. Incluye en el presente trabajo la historia completa de la familia aludida con los datos de anatomía gruesa y microscópica referentes a los individuos atáxicos y a los normales que sirvieron como tipo de comparación. Los cambios encontrados en el sistema nervioso central consisten principalmente en una reducción del tamaño del cerebro y médula espinal, especialmente en el cerebelo y las partes directamente relacionadas con él. Esto puede considerarse como una hipoplasia o inhibición del desarrollo del sistema propioceptivo, parte del sistema motor y algunas de las estructuras que unen a la médula oblonga con el cerebelo, la cual tiene lugar durante el crecimiento, con una degeneración apenas marcada o aumento secundario del tejido neuróglia. Después de revisar someramente la ataxia de Friedrich y la hérédito-ataxia cérébelleuse de Marie, el autor interpreta la condición de las aves examinadas como una combinación de las dos afecciones humanas mencionadas.

Translation by José F. Nonidez  
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From an egg produced by the weakening influences of 'reproductive overwork' a female pigeon no. 151 was hatched in 1914 which showed a marked lack of power over the voluntary movements of the head and body. This lack of coordination was practically completely lost in the adult bird. The affected female was bred to two normal males, A126 scraggly and C-B9. The derangement has been inherited through four generations descended from either male.

The parents of no. 151 were raised by Professor Whitman. The male parent, a two-banded homer H-A, had no ataxic symptom nor did his sire or dam. H-A homer was an inbred, for its parents were brother and sister. The dam of no. 151 was of the homer-carrier type, normal and without ataxia. These parents of no. 151 laid for the last time in 1914 on about October 12th to 14th, and one of these eggs hatched the ataxic female. This female (no. 151) was thus hatched at the end of the season from a pair of birds which had been kept constantly at work and from parents one of which was an inbred.

When first out of the nest the abnormality of no. 151 was noted, and therefore the next pair of eggs produced by parents of no. 151 were also incubated. The two birds hatched from these eggs resembled no. 151, but were not ataxic. There is no record of ataxia in any of the other descendants of the parents of no. 151 during the entire previous four years. There is reason to believe that this character arose within the germ that produced no. 151 and that the weakening effects of abnormally rapid egg-laying and possibly the inbreeding of the male parent were causally related to the appearance of the character.

The sire (A428) of the 'scraggly' male no. A126 was a checkered *Columba livia domestica*, which had the tips of the wings white. As is well known, white is apt to appear in these outmost wing-feathers in many breeds of the domestic pigeons. This restricted

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Male C-B9, with which the ataxic female (151) was mated for a short period prior to her mating with the scraggy male, was a pure wild rock pigeon (*Columba livia*). It was hatched in 1910 from parents obtained (1908) from the caves of Cromarty, Scotland. The three offspring of this very strong and vigorous male and the ataxic female were normal in appearance and behavior, but in the next generation a portion of the offspring exhibited ataxia. No white color has thus far appeared in any of their descendants.

Ataxia, scragginess, and white color have all appeared in three generations derived from the mating of the scraggy male and ataxic female. Without here entering into full considerations of the proportions of abnormals to normals for each of these three characteristics in the different generations, it can be said that the first generation showed relatively few abnormals—ataxics, scraggles, or whites. Later generations have shown higher proportions of affected individuals, and the combination of ataxia and scragginess has there been obtained.

The ataxia of the original ataxic bird (no. 151) disappeared some time after she became adult. When she died recently, she seemed quite normal. This is not true of many or most of later ataxics, which show much more extensive lack of coordinations, and maintain them till the end of life. Of course, the extreme ataxics do not live long. Doctor Riddle describes the scragginess as follows. This, he says, is a plumage defect, the feathers lack barbules and hooklets, and as a result the barbs of all feathers of all these birds hang loosely apart so that the wing feathers give no resistance to the air, and the birds cannot fly. Such feathers present a very peculiar and bristling appearance.

The statement concerning pedigree, and behavior of each of the four birds, which were sent us runs as follows:

No. K137. Young of cage 131. Second generation hybrid (not counting original ataxic and scraggy as first generation). Parents: male A456 and female A446 (neither of which showed ataxia or scragginess). The parents of these latter: original ataxic female 151 and original scraggy male A126, from eggs laid 4/8/17. Ataxic—gait unsteady, flies very little or not at all, tips backward, and also tends to tip sidewise.

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on one side of the body with the wall of the pen. If food or water is placed in the middle of the cage on the floor, they have great difficulty in reaching it. Food is really the only thing which will make them attempt to walk, except when they are frightened or excited. In their attempts to walk they fall forward or sideways or just stumble along reeling like a drunken man, 'démarche ébrieuse'. When they fall forward they try to get up with their bills against the floor pushing back the body and flapping the wings with much effort. When they fall to the side they usually roll over once or twice. Sometimes they fall to the right, while at other times they fall to the left and then roll until they reach some obstruction which helps them to get up with the aid of flapping the wings. Flying is practically impossible in all birds, if they are thrown free in the air, they flap their wings irregularly and cannot fly above the height they are thrown, but go directly down to the floor notwithstanding that their flapping efforts are much more intense than those of normal birds. When the birds are excited or frightened, the disturbances of the irregular movements stated above are much more apparent. Such a movement as the so-called "tremulance" or oscillatory movements cannot be observed either when the birds are excited or at rest. Ocular movements are free, no deviation and no nystagmoid jerking can be substantiated. The reflexes which may be elicited from the cornea are normal. When they are put on a rotating chair they show the head nystagmus characteristic of normal pigeons. If they are rotated more than five or six times they lean against the cage wall or lie down, exhibiting regular head nystagmus. When blindfolded the birds reveal no increase of the disturbances of coordination.

As far as can be determined, sight and hearing are normal, the birds can recognize food and an observer who may be approaching, they also react to a sudden sound by raising the head and trunk suddenly, but immediately lower them again. Pupils are equal and react to light promptly. The sensibility to touch as well as to pain appears unaffected in the skin, the birds react to stimulation with direct movements, but all these movements are quite sluggish. The toes of three affected pigeons are more or

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